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THE AUTONOMIC PHARMACOLOGY OF THE GASTRIC JUICES*

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WE are presenting the following experiments because they have brought to light important new phenomena concerning the chemical activities of the stomach, especially as related to autonomic pharmacology. We do not intend in this presentation to enter into any elaborate theoretical discussion, since it does not seem to us that the present accumulated facts warrant generalizations.

In many respects the results we have obtained by human experimentation quite definitely contradict the rather chaotic results that have largely been built up by animal experimentation. The difficulty with translating the results of animal experimentation to human physiology are too well known to need elaboration. Fundamentally, the anesthesia and the emotional state the animal is thrown into by the experimentation, as well as the fact that various species differ from one another in important fundamentals of physiologic reaction, make the human application of these results precarious.

We believe our results to be quite valid because the experiments were carried out on human beings without any anesthesia and without any mutilation, such as is involved when sympathetic or parasympathetic fibers are cut, electrically stimulated or surgically handled in any way. The procedure in general has been to select patients with mental disease who are physiologically healthy so far as we can discover by any technics of the present day. Mainly male cases of dementia praecox have been used that are known to us as to hematology, tuberculosis, syphilis, infection in general, blood pressure, heart, kidney disease and basal metabolism. In fact, these individuals have been more conclusively shown to be physiologically healthy than most of the so-called healthy subjects selected for experimentation in other clinics and by other investigators. Moreover, these patients are exceedingly docile; they submit themselves for experimentation time after time, during many years of study.

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In this series of experiments we have balanced certain of the so-called parasympathomimetic and sympathomimetic drugs against one another, following along a line of experiments we have utilized in evaluating and attempting to understand blood pressure, gastrointestinal motility and other physiologic functions. The parasympathomimetic drug used was acetyl-beta-methylcholine chloride (mecholyl); the sympathetic stimulant was beta-phenyl-isopropylamine sulfate (benzedrine sulfate). We have used atropin to check and inhibit the effects of mecholyl and, because it removes the influence of the parasympathetic nervous system, to enhance the effects of benzedrine. Since the esterases of the body inhibit acetylcholine and thus inhibit the action of the parasympathetic nervous system, prostigmin and physostigmin, which inhibit the esterases, have been used to enhance the effect of mecholyl or to obtain parasympathetic stimulation. We fully realize that none of these drugs have any "pure" action and possibly it would be better to drop the terms parasympathetic and sympathetic entirely in the discussion of these experiments and speak of the effects of mecholyl, benzedrine, atropin and, as a group, prostigmin and physostigmin on the gastric secretions. The fact is, however, that irrespective of theory we have been able to change the quantity, alkalinity and acidity, pepsinogen content and mucin content of the stomach at will and consequently feel that our results add much that is striking and new in human physiology.

Elsewhere[1] we have shown that benzedrine relaxes gastric spasm. In a publication to appear shortly[2] we show that mecholyl rapidly and measurably increases the tone of the gastrointestinal tract. We have followed these facts as to tonus into the following experiments on stomach secretions.

PROCEDURE

The patients came to the laboratory in the morning after a 16 hour fast. The gastric juice was obtained by means of a Levin's nasal tube, which was readily inserted with the patient in the prone position. Uncooperative patients were not used; in general it was found that after one or two experiments our patients made ideal subjects for gastric intubation.

The whole amount of fasting juice was obtained

by repeated aspirations over a period of about 20 minutes. The tube being left in situ, an injection of one or more of the various drugs was then made subcutaneously. In a few experiments drugs were given by mouth. The following drugs were used: mecholyl (acetyl-beta-methylcholine chloride)*, benzedrine sulfate (beta-phenyl-isopropylamine sulfate)†, atropin sulfate, physostigmin and prostigmin‡. These were given either singly or in various combinations according to the type of experiment planned. The dosage usually used was as follows:

Mecholyl	25 mg.
Benzedrine sulfate	40 mg.
Atropin sulfate	1.3 mg.
Physostigmin	0.65 mg.
Prostigmin	0.5 to 1 mg. (1-2 cc. of 1-2000 solution)

Following administration of the drug, gastric juice was aspirated at frequent intervals—every 2, 5 or 10 minutes, according to the aim of the experiment. Test meals were not given because we wished to obtain a secretion picture of these chemicals that was free from the physiologic reactions of digestion itself. This is in line with the work of Bloomfield and Polland[3] on histamine. As much gastric juice was aspirated as could be obtained and the volumes recorded. The presence or absence of free hydrochloric acid was immediately tested for by Congo red paper. Litmus paper was used as a rough qualitative test for total acidity or alkalinity. Free hydrochloric acid was determined by titration with N/10 sodium hydroxide, using Töpfer's solution as an indicator. Total acid was determined by further titration, using phenolphthalein as an indicator. Combined hydrochloric acid was determined by the method of Michaelis. The total hydrochloric acid was in most cases substantially the same as the total acid.

In those samples of gastric juice in which free hydrochloric acid was absent the degree of "acid deficit" was obtained. This was tested by titration with N/10 hydrochloric acid, using Töpfer's solution as an indicator, hydrochloric acid being run in until the typical red color was obtained. The pH of the gastric juice was measured electrometrically in typical experiments using the Beckmannmeter.

Chlorides were analyzed in several cases by the Wilson and Ball method.[4] The pepsinogen content of the juice was determined in typical experiments by Mett's egg albumin tubes, using successive dilutions of gastric juice (1-2, 1-4, 1-8, 1-16), alkaline specimens being adjusted to the proper pH for peptic activity. Rennen determined in typical cases was found to be parallel with pepsinogen. The mucin content was estimated mostly by the presence or absence of a viscid appearance in the juice and by testing for some of the chemical manifestations of mucin, that is, alkalinity and a cloudy appearance with weak acids, such as dilute hydrochloric or dilute acetic acid.

A few control experiments showed that the presence of a tube in the stomach for the period of a typical experiment in itself produced no essential or comparable changes in quantity or chemistry of the stomach juices.

MECHOLYL

When this chemical is given subcutaneously in the dosage of from 25 to 40 mg., certain general effects, which have been fully described in the literature and to which we have paid attention in a previous publication,[5] become quite marked—flushing of the face, neck, chest and back; sweating, which soon becomes profuse and alkaline; lacrimation; rhinorrhea; salivation; rise in pulse rate and fall in blood pressure.

In the gastric contents the following effects were regularly and invariably noted: (1) a marked increase in volume, (2) a great increase in mucin, (3) a sudden reversal within 2 to 8 minutes of the acid reaction to a definitely alkaline reaction, (4) disappearance of free hydrochloric acid with the production of a marked "acid deficit", (5) reduction in the combined acid and (6) disappearance of pepsinogen and rennen.

Chart 1 is that of a typical experiment and speaks for itself in respect to the quantitative and qualitative changes noted in the gastric juice. The sharp drop from acidity to alkalinity is graphically shown. The increase in quantity is, as will be noted, nearly double. What is not indicated by the chart is the marked and sudden appearance of mucin in large quantities and the complete disappearance of pepsinogen.

If we divided up the time of the experiment into the stimulation phase, during which time the mecholyl acts, and the recovery phase, at which time the gastric juice returns to normal, the various factors in the gastric contents from a group of ten cases may be analyzed as follows:

Quantity. Fasting Content. This varied from 16 to 182 cc.

Stimulation Phase. This lasted from 15 to 95 minutes, average 35 minutes, during which time both the full systemic effects of the drug and the striking effects on gastric juice were demonstrable. The total volumes varied from 78 to 350 cc. Secretion per minute varied from 3.1 to 12.1 cc., average 6.7 cc.

Recovery Phase. This lasted from 20 to 85 minutes, average 52 minutes. The total amount of juice removed during this time varied from 14 to 161 cc., the secretion per minute being estimated from 0.3 to 4.8 cc., average 1.9 cc.

Free Hydrochloric Acid. Fasting Content. Free hydrochloric acid varied from 8 to 68 units (cc. of N/10 sodium hydroxide per 100 cc. gastric juice). In one case, achlorhydria was present (acid deficit—18 units) and there was no appearance of hydrochloric acid following histamine stimulation.

Stimulation Phase. A striking diminution in free hydrochloric acid occurred in every instance, regularly beginning within the first 4 minutes. In one case in which continuous aspiration was done and the time recorded by stop watch, the disappearance of free hydrochloric acid occurred in 2 minutes, 37 seconds. The acid deficit reached its maximum between 5 and 15 minutes, average 10 minutes.

*Furnished through the courtesy of Merck & Co., Inc., Rahway, N. J.

†Furnished through the courtesy of Smith, Kline & French Laboratories, Philadelphia, Pa.

‡Furnished through the courtesy of Hoffmann-La Roche, Inc., Nutley, N. J.

Recovery Phase. Acidity gradually returned to normal in most instances; in a few cases the hydrochloric acid content was greater than that in the fasting juice (overcompensation). Free hydrochloric acid in the recovery phase varied from 2 to 50 units.

The beginning of acid return varied from 14 to 98 minutes, average 41 minutes. In one case (H. H.) in which the fasting contents showed 8 units of free hydrochloric acid, there was no return of acid at the end of 1 hour, 35 minutes. In another case (E. D.), with achlorhydria of the fasting contents, hydrochloric acid did not appear at any time during the experiment.

It should be noted that during the height of reaction there is a definite decrease in chlorides per 100 cc. of gastric juice.

Total Acidity. This was practically coincidental with total free hydrochloric acid. The titration of an anacid gastric juice, consisting essentially of mucin, is extremely difficult and subject to error. We felt that the pH determinations were more satisfactory.

Mucin. This is the most striking reaction. During the stimulation phase, the mucin completely dominates the visible picture of the gastric juice. The clear gastric juice becomes cloudy and viscid during the stimulation phase and then quickly becomes clear again in the recovery phase.

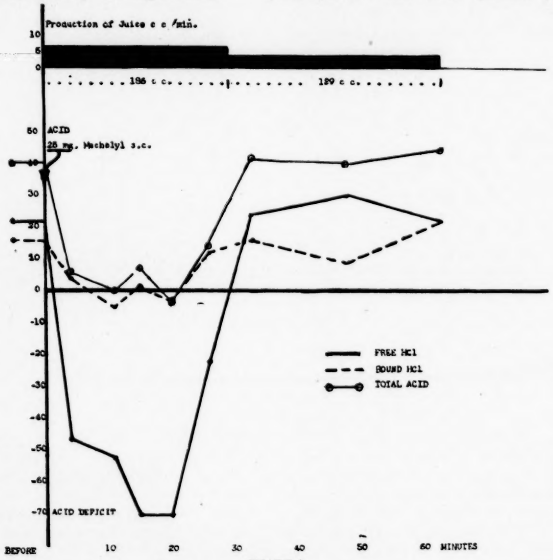

CHART 1.
Effect of Mecholyl on Gastric Juice.

Hydrogen Ion Concentration. The pH was determined in 2 typical cases. This corresponded in all respects with the qualitative reactions obtained with litmus and Congo red and with the titration results.

The pH increased in one case from 1.9 to 8.9 in the stimulation phase (15 minutes), returning in 33 minutes to 1.7; in the other case it increased from 2.56 to 8.95 in the stimulation phase (11 minutes), returning to 1.99 at the end of experiment (42 minutes).

Chlorides. These were determined in 3 cases and showed the following results:

	Before	Stimulation Phase	Recovery Phase	
(1)	485	438	484	mg./100 cc.
(2)	597	402	514	"
(3)	679	641	676	"

Pepsinogen. This was determined in 9 cases. There was always complete disappearance of pepsinogen during the stimulation phase, the pepsinogen returning to normal in the recovery phase, corresponding in all respects with the disappearance and reappearance of free hydrochloric acid.

The outstanding gastric effects of mecholyl are that the activity of the acid glands ceases entirely and mucin stimulation reaches an extraordinary maximum within a short period of time. It is interesting to note, and we believe it to be important, that the intradermal injection of mecholyl probably gives a more vigorous

reaction than the subcutaneous use of the drug. Whenever mecholyl is given, even in minute doses, the trend is in the same general direction, that is, a diminution or disappearance of the digestive secretions, hydrochloric acid and pepsinogen, and finally the appearance of mucin.

Iontophoresis of Mecholyl. Very interesting and corroborative is the result of iontophoresis. This experiment which is to introduce mecholyl ions into the body by the positive pole of the galvanic current was carried out as follows: a large electrode moistened with 5 per cent mecholyl solution was placed over the abdomen with the negative electrode on the patient's back. Ten milliamperes of current were used. After the lapse of 10 minutes the gastric juice, previously normal, turned alkaline and contained a considerable amount of mucin throughout the time of active iontophoresis. These changes disappeared when the experiment was completed and the gastric juice returned to its normal condition. We are not yet ready to report in any further detail on this experiment which will be the subject of a future communication. The general effects during the time of iontophoresis were relatively mild; blood pressure and pulse rate hardly changed. There was some degree of sweating and flushing but the patient was not, so far as we could learn, uncomfortable.

BENZEDRINE SULFATE

The administration of benzedrine sulfate subcutaneously in the usual dosage of 40 mg. produced typical results. Though little general visible reaction took place, there was a definite rise in blood pressure associated, usually, with some slowing of the pulse rate. A comparison of the benzedrine results on the gastric secretion with those of mecholyl stimulation shows a striking contrast (chart 2). In general it may be stated that the digestive juice elements increased, that is, there was increase of free hydrochloric acid, total acids and pepsinogen. There was no mucin production. The quantity was not noticeably altered by benzedrine stimulation, but, if any change was present, it was in the direction of a slight diminution of juice. In most respects benzedrine stimulation resembles that of histamine except that the quantity is not increased. In contrast with histamine the patient felt no ill-effects during the experiment.

An analysis of the results on gastric secretion following the administration of benzedrine sulfate to a group of cases is as follows:

Quantity. Fasting Content. This varied from 12 to 178 cc.

Stimulation Phase (when acidity was either increasing or remaining at same level). The volumes collected during 43 to 110 minutes varied from 36 to 150 cc. and the secretion rates per minute were 0.5 to 2.7 cc., average 1.4 cc.

Recovery Phase. This was difficult to determine. We are not able to give exact pictures, but believe that there was no essential difference in quantity.

Free Hydrochloric Acid. This always became increased.

Fasting Content. In 6 cases that showed free hydrochloric acid the range was from 24 to 62 units.

Stimulation Phase. In 3 cases an increase in free hydrochloric acid was noted: from 38 to 64 units in 1 hour, 20 minutes; from 24 to 66 units in 26 minutes; from —12 to +8 units in 61 minutes. In 3 other cases, in which only the pH was determined, the results were as follows:

(1) 1.6 to 1.2 in 25 minutes
(2) 1.6 to 1.5 in 13 minutes
(3) 1.7 to 2.6 in 69 minutes.

In 4 cases with achlorhydria (not pernicious anemia) the fasting hydrochloric acid deficit was from 12 to 64 units. The hydrochloric acid

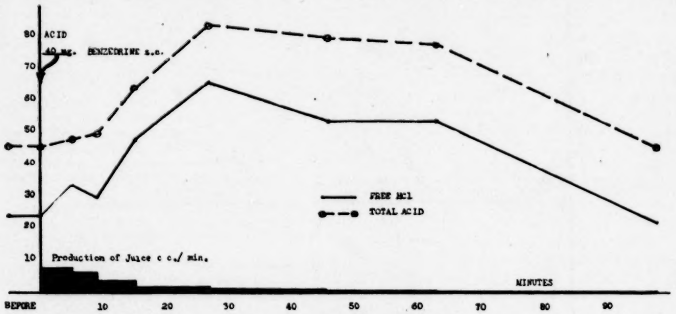

CHART 2.
Effect of Benzedrine on Gastric Juice.

assumed in advance that if given before the administration of mecholyl, the mecholyl effects would not appear. This was in part confirmed by our experiments and in part contradicted. The amount of secretion was not increased by mecholyl administration after atropin; the diminution continued. However, the free hydrochloric acid, which was +64 units at the beginning of the experiment and which remained at about that level after 2.6 mg. atropin had been administered, dropped to +25 units after the administration of mecholyl and remained at this level for 1 hour and 55 minutes. It should be noted that a tremendous amount of atropin was given. In other words, while the secretion was not increased by mecholyl, the mecholyl effect on the acidity occurred though in much reduced measure.

sulfate would enhance the benzedrine reaction, since by removing the parasympathetic brake or effect of acetylcholine, the sympathetic stimulation would be increased. This occurred in our experiments. A typical chart is shown (chart 4). The increase in acidity reached a higher level than in any other of the experiments, and the juice continued at this heightened level of acidity for a longer period of time. The amount of secretion remained fairly consistent throughout the experiment. Thus, in the 40 minutes following the administration of benzedrine, the chart shows by the thickened base line that the secretion per minute scarcely changed at all and certainly was not diminished by the atropin. This is definitely in harmony with our results on blood pressure. In large doses, benzedrine sulfate raised the blood pressure. If atropin is added to the benzedrine, the rise becomes very marked.

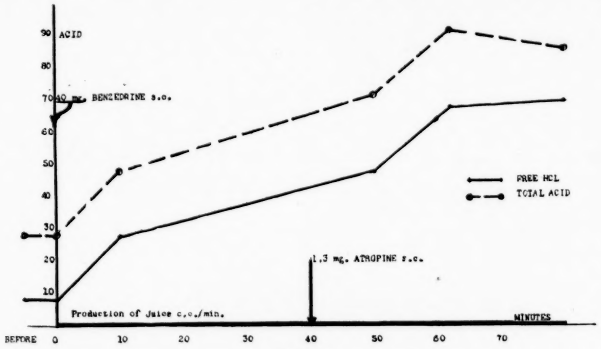

CHART 4.
Effect of Benzedrine and Atropin on Gastric Juice.

Mecholyl Followed by Atropin. The results of the administration of mecholyl followed by atropin are graphically recorded in chart 3. It may be seen from this chart that when the characteristic effects of mecholyl were produced —achlorhydria with acid deficit, increase in volume per minute—atropin was given in two divided doses of .65 mg. each. Clinically, this was followed by a striking and prompt disappearance of all external effects, as noted in our previous experiments. In the gastric juice, the acid deficit became lessened, so that within 10 minutes it had returned to almost 0 (—5). This was comparable to the previous experiments with mecholyl alone. The total acidity, reduced to 0 with mecholyl, gradually rose to normal. The amount of juice produced became strikingly reduced so that in 1 hour only 15 cc. were collected.

Benzedrine Sulfate Followed by Atropin. It would be expected, theoretically, that the administration of atropin following benzedrine

Benzedrine Sulfate Followed by Mecholyl. In previous experiments it was noted[6] that when benzedrine effects were obtained, the injection of mecholyl completely abolished them and then mecholyl effects followed as if no benzedrine had been given. This was true in the case of blood pressure and heart rate. It was equally true of the gastric juice, as is strikingly shown in chart 5. This chart shows that after benzedrine the rise of the free hydrochloric acid was from 4 units to 25 and that the amount of secretion per minute was about 2 cc. When the mecholyl was administered, there was an immediate drop in the acidity to —50, with a final deficit of 58 units. The volume increased to 6 cc. per minute. The hydrogen ion concentration which had increased to pH 1.8 at the height of the benzedrine reaction decreased to pH 9.0 at the height of the mecholyl reaction. Mucin appeared in large

quantities during the mecholyl phase and disappeared at the end of 40 minutes, when the gastric juice became somewhat more acid than it had been during the height of the benzedrine reaction. In this experiment it will be seen that the parasympathetic stimulation predominated over the sympathetic stimulation or, to put it chemically, the mecholyl effect was greater than the benzedrine effect. This has been uniform in our experimentation.

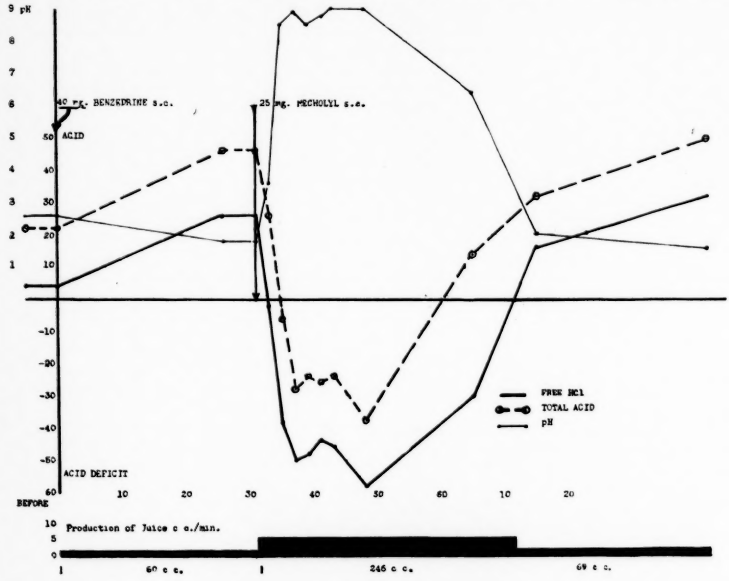

CHART 5.
Effect of Benzedrine and Mecholyl on Gastric Juice.

Prostigmin (or Physostigmin) Followed by Mecholyl. Theoretically it would be expected that the use of both prostigmin and mecholyl would result in an increased general parasympathetic effect, since prostigmin is one of the group of drugs that is supposed to inhibit the esterases, which, in their turn, destroy or inhibit the effects of the bodily acetylcholine or the acetylcholine group of drugs. This theoretical expectation was completely confirmed in all of our experiments, some of which will be recorded in a later publication on sweating and other physiologic processes. Chart 6 shows graphically this relationship. The pH started at 2.1, a typical acid gastric juice, and then dropped spontaneously to just under 2. One cc. of prostigmin given subcutaneously caused a slight rise in the pH, that is, a slight diminution of acidity, and, in 16 minutes, 10 mg. mecholyl were given. This dose by itself in the same patient was unable to change the pH in any great measure. In this experiment the 10 mg. of mecholyl changed the pH from 2.6 to 7.5, at which level the gastric juice remained for about 15 minutes and then dropped within

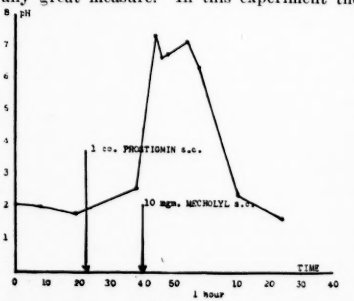

CHART 6.
PH of Gastric Juice Following Administration of Prostigmin and Mecholyl.

the next 30 minutes to its original acidity. In other words, prostigmin acts as a synergic factor for mecholyl, thus enabling small doses of mecholyl to produce a marked effect in changing the gastric juices from acid to alkaline. This relation we believe to be of importance in the iontophoresis treatment of ulcers, arthritis and tachycardia—in other words, those clinical uses to which mecholyl has already been put. We may state at this point that physostigmin has fundamentally the same effect as prostigmin in relation to the acidity of the gastric juices and the amount of gastric content. By itself it produces a slight change in the direction of alkalinity, that is, it will reduce the acidity but it will not produce an alkaline secretion or mucin. If, however, given prior to the administration of mecholyl, a small dose of mecholyl will produce drastic changes in the gastric juices of the nature here described as the typical mecholyl effect.

LITERATURE

There is no literature so far as we know that concerns gastric secretion following the use of benzedrine, physostigmin, prostigmin or the combinations that we have discussed. We may state that our work was undertaken without knowledge of any previous experimental work on man.

Mecholyl has been studied in relation to its effects on the gastric juices by Ferguson and Smith[7] in respect to the gastric acidity of monkeys. Their technic involved the use of a test meal. They found that the free gastric acidity was completely abolished if a sufficient dose of mecholyl was given, and their results with atropin run parallel with ours. They found that the total acid was parallel with the free acid curve. The total chlorides were unaffected. The mucous secretions were definitely increased, largely in the saliva. However, in human experimentation, Abbott[8] reported results that are opposite to ours. His conclusion was that mecholyl increased the acidity, at the same time increasing gastrointestinal tone and motility. In criticism of his results it may be stated that he probably used too small a dose. He gave 5 to 10 mg. subcutaneously and 200 to 300 mg. by mouth. Oral administration brings practically no results, as we learned by repeated experiments. Moreover, the technic of his experimentation was erroneous in that the first sample that he took after the administration of the drug was obtained in 5 to 20 minutes. We have experiments on record in which the mecholyl effect took place and disappeared completely within the same period of time, especially with small doses such as he administered. In other words, he did not produce the full mecholyl effect in his experiments and his technic of observation missed the changes that took place.

Since 1926 the synergic relation of physostigmin and acetylcholine derivates has been known. The literature, while scanty, is authoritative and starts with the work of Loewi[9, 10] himself. It was he who first discovered the production of acetylcholine by the parasympathetic nervous system and who later showed that the esterases have a destructive enzymic effect on this chemical and that physostigmin inhibits the destructive power of the esterases. Hunt,[11] Führer,[12] and Freud and Uyldert[13] have confirmed this general relation.

DISCUSSION

From the clinical standpoint, many possibilities suggest themselves, for example, "functional" or "false" achlorhydria may be indicative of a vagal or parasympathetic state, more exactly an overproduction of acetylcholine, and so may an excessive mucin concentration in the gastric juice. Hyperchlorhydria may be evidence of sympathetic overstimulation or its chemical equivalent. The lack of mucin in the gastric juice of certain individuals may also point in this direction. To speculate further, the hyperchlorhydria of peptic ulcer and the associated diminution in mucin content, as demonstrated by Anderson and Fogelson,[14] may be based upon an increased activity of the sympathetic nervous system.

Therapeutically, measures for raising the mucin content of the gastric juice and lowering the acidity have been found to be of distinct value in peptic ulcer. Mucin has been utilized in this respect with some success. This might be achieved without the feeding of mucin by the use of various parasympathetic stimulants, such as mecholyl, physostigmin or prostigmin. Functional achlorhydria and gastric symptoms associated with a possible "vagal" state might be benefited by the sympathomimetic drug, benzedrine sulfate. The diagnostic and therapeutic implications of exact knowledge of the effects of these drugs on the gastric juice are seen to be great and should repay further study and clinical investigation.

SUMMARY OF RESULTS

The effects of certain drugs upon the gastric secretions are here presented. These effects may be summarized as follows:

(1) Mecholyl remarkably stimulates the production of mucin, regularly increases gastric juice secretion and inhibits the production of hydrochloric acid and pepsinogen, thus producing a copious alkaline gastric juice.

(2) Benzedrine sulfate either slightly diminishes or does not affect the amount of gastric juice, but definitely and regularly increases

the free hydrochloric and the pepsinogenic activity.

It is noteworthy that mecholyl also increases gastrointestinal tonus and benzedrine sulfate relaxes or diminishes spasm.

(3) Atropin regularly diminishes the secretion of gastric juice, without, in any noticeable way, affecting the chemical relationships.

(4) Prostigmin and physostigmin produce or tend to produce an alkaline gastric juice of the same general nature as the mecholyl effect, but in lesser degree and without noticeable general bodily effects.

(5) Atropin given in advance of mecholyl prevents its effects in so far as increase in rate of secretion is concerned and diminishes its effects in so far as alkalinity is concerned. It has the same general relation to the mecholyl effect given after this chemical in so far as quantity is concerned, but seems to have no definite effect on the alkalinity produced by mecholyl.

(6) Mecholyl completely overshadows the benzedrine sulfate effect, but after its effect has worn off the benzedrine sulfate effect becomes again manifested.

(7) Atropin administered after benzedrine sulfate enhances its general effects on the gastric juice.

(8) Prostigmin and physostigmin act synergistically with mecholyl on the gastric juices, thus bringing it about that a small dose of mecholyl will produce a complete mecholyl effect if either of these drugs is administered previously or concurrently.

(9) Iontophoresis of mecholyl produces definite mecholyl effects on the gastric juice.

It appears to us that these experiments, especially those in which alkalinity is produced, should receive consideration on the part of clinicians for the treatment of gastric ulcer and hyperacidity. We believe that the iontophoresis method, if carefully worked out, may furnish a valuable way of administrating these drugs so that a moderate alkalinity, with mucus, is maintained over a considerable period of time.

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FRACTURES IN THE NEWBORN*

A Plea For Adequate Treatment

BY AUGUSTUS THORNDIKE, JR., M.D.,† AND F. RICHARD PIERCE, M.D.†

THE problem of adequate treatment of birth fractures commands the interest of the obstetrician, pediatrician, surgeon, and orthopedist. The old teaching that mere fixation is the only treatment required to produce good results in this class of fractures is no longer considered satisfactory. The general improvement and interest in fracture therapy today, largely stimulated by the Fracture Committee of the American College of Surgeons, demand that even these specialized fractures must not be neglected.

In a review of the last six thousand fractures at the Children's Hospital, Boston, it was a revelation to find cases of birth fractures that had come to the hospital at twelve to eighteen months of age because of unilateral bowlegs. Needless to say, inadequate treatment of birth fracture is one explanation of such a condition. The importance of proper treatment of these fractures must not be overlooked.

We feel that in the great majority of cases the attending physician cannot be blamed for a fracture occurring during the course of delivery. We do not agree with Newman and Levy[1] that poor technic and injudicious use of instruments are the chief causes of birth fractures, but believe that they are accidents and that as such are many times unavoidable.

Just as remarkable in the accidents of labor are those cases of precipitate labor which are born without injury. Von Winkel[2] reports two hun-

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dred and sixteen cases of precipitate labor without a single instance of serious injury. Strumpf[3] quotes Sir William Osler as follows: "The child was born in the toilet of a fast-moving train, fell to the tracks and showed only slight contusions." With such reports as these, one might wonder why the incidence of birth fractures was not greater.

Reported statistics on the incidence of birth

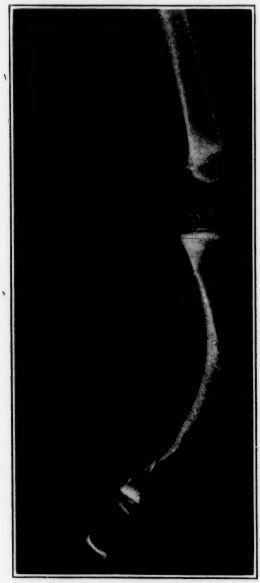

ILLUSTRATION NO. 1.
X-Ray No. 42259—Old Fracture of Tibia.
Unilateral bowleg from untreated birth fracture of the tibia.

fractures vary from 39 cases in 33,000 deliveries reported by Truesdell[4] to 3.6 per cent—7.7 per cent femur fractures in breech deliveries by Ehrenfest.[5] It is generally agreed as Gibberd[6] has pointed out, however, that there is a higher incidence in all types of birth injuries in breech presentation. One factor is self-evident in reviewing the literature and that is the paucity of reports and studies of larger series of cases.

In this series of 115 consecutive cases of birth fracture reviewed at the Children's Hospital, Boston, 53 fractures of the clavicle, 38 of the humerus, 20 of the femur, 4 of the tibia including 3 with fibula, have been studied. No fracture of the radius and ulna was found in this collection of cases, and cases of fracture of the skull or spine have not been included. Furthermore, 20 per cent of the fractured clavicle cases, 92 per cent of fractured humerus cases, 72 per cent of fractured femur cases, and 50 per cent of fractured tibia cases required unusual obstetric maneuvers for the delivery of

ILLUSTRATION NO. 2.
X-Ray No. 162625.
Seven months of age. Unrecognized birth fracture. Lateral bowing with shortening.

a live baby. With the revelation of these figures, blame for such accidents cannot justly be placed solely on the medical man in attendance at the time of delivery.

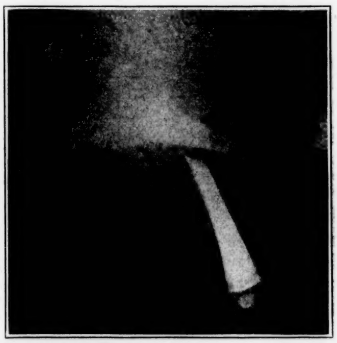

ILLUSTRATION NO. 3.
Case No. 129415—Fractured Femur.
Fractured left femur on entry. Displacement and overriding present. Ten days of age on entry. Callus prevented proper realignment.

However, 40 fractures, or 34 per cent, of the entire group were not recognized at the time of their occurrence, a situation for which the attending physician or obstetrician may be con-

	Name and Birth Wt.	Hosp. No.	Age	Sex	Labor and Type of Delivery	Location and Type	Time of Recognition	First X-Rays	Early Treatment	Late Treatment	Complications	Follow-Up	Remarks
1.	J. M. 6	115576	1 day	M	Cesarean	Rt. femur oblique mid and upper 1/3 posterior displaced lower fragment marked overriding	Birth	1 day	Leg strapped to body. Position unsatisfactory. Shifted to post. wire splint—position still not good. Put on Bradford frame with overhead traction 3 lbs.	Not seen after discharge	None	1 O.P.D. visit. Last x-rays at 1 mo. Good position at that time.	On overhead traction 12 days. In hospital 1 mo. Gained 1 lb. 3 oz.
2.	D. R. 7-8	109058	14 mos.	F	Breech	Marked ant. bowing due to old healed fracture	2 wks. By District Nurse	2 wks. by attend. phys.	None	Thomas heel 1/4" shortening at 3 yrs. of age	Shortening	Yes. Heels and pads fitted, etc.	Recognized at 2 wks. by attending physician. Callus at that time
3.	M. A. 9+	113042	1 yr.	F	Breech Difficult	4 cm. from upper end of lt. femur. Sharp lat. angulation.	1 yr.	1 yr.	None	Plaster boot valgus pos. Phy. Tr. Thomas heel later	Unilateral bowed leg	Yes—fracture straightened with changing position by cast	Not recognized by attending physician
4.	M. A. 8-6	105266	2 wks.	F	Primip. Breech	Transverse midshaft. Post. displacement of lower fragment overriding 1/2 cm.	At birth	12 hrs.	Post. wire splint 14 days then coaptation splints	None	None	Yes. O. K. in 1 month—splints off	
5.	M. C. 8-3/4	129415	1 wk.	F	Breech Leg snapped in attempting to twist body	Transverse mid and upper 1/3. Ant. displacement of upper 1/3 with overriding.	At birth	1 wk.	Overhead traction 3 lbs. 17 days.	None	None	10 mos. later O. K. except for slight bowing	This case re-x-rayed at frequent intervals for record
6.	B. H. 7	159443	2 days	F	Footling trauma to rt. leg	Chip fractures off the lower margins of both femora	1 day	2 days	Not admitted	None	10-15° flexion contracture at 6 mos.	Yes—till 6 mos.	
7.	B. F.	167497	2 days	F	26 hr. labor. Breech delivery difficult	Rt. oblique mid 1/3 with anterior angulation	Birth	2 days (34713)	Traction with light bed 2½ rt. (overhead) 20 days	None	None	Yes—1 wk.	Good result.
8	M. A. W. 10	145297	1 mo.	F	Difficult version breech delivery	Upper of lt. Femur	1 month not rec. by attending physician	About 1 wk. outside. 1 mo. at this Hospital.	None	None	None	Yes	Good result. Mother told on discharge from Maternity Hosp. that child did not move leg. Fracture not diagnosed until at C. H.
9.	L. C. 5-11	181394	2 wks.	F	Forceps delivery	Midshaft right	Birth at Maternity Hospital	Birth	Bandaged lt. femur to abdomen	None	Child also had club feet, club hands, left torticollis, micrognathia, flexion contracture of all joints, fracture lt. humerus	None	?
10.	H. K. 6-7	184061	13 days	M	Cesarean Difficult	Oblique midshaft	Birth	10 days	Wire splint	Overhead traction at 13 days for 10 days	None	None	Good result.
11.	H. K. P. 9	147824	3½ mos.	M	Cesarean section difficult	Fracture of neck of left femur	3½ mos.	1 mo. outside	None	Putti pillow 1 mo. then at 4 mos. abduction splint	Sl. limitation of abduction. Rt. has marked ant. bowing of upper 1/3. 3/8" shortening osteotomy at 10 mos.	Lt. leg 3/8" longer than rt. Mod. pronation of both feet. Child didn't walk until 2 yrs.—was mentally definitely retarded.	Classical signs present fracture not diagnosed by attending physician although in letter he stated x-ray was taken.
12.	W. P. 6-3	172441	16 mos.	M	Normal	Lt. with lateral bowing	Birth (36963)	16 mos. C. H.	Plaster 1 wk. Bandage 2 wks.	None	.5 cm. shortening	Deformity corrected once—given heel to correct shortening. Walked at 1 yr.	
13.	B. A. 9	169673	9 days	M	Difficult Breech	Midshaft left	7 days x-ray at Maternity Hosp.	9 days at C. H. (35804)	Splint 7-9 days. Lat. bowing with angulation and 1 cm. overriding on entry. Splint failed to hold properly.	9-26 days put in traction (overhead)	None	Yes	Good result.
14.	P. A. 6-¼	168490	11 mos.	F	Normal	Old midshaft healed	At birth	16 mos. at C. H.	None	Shoes built up	Shortening	No	" Permanent leg shortening.
15.	M. M. 8	172150	3 wks.	F	Instrumental breech	Mid and upper 1/3 rt. backward displacement (on entry to C. H.)	Birth by attending physician	By attend. phys. 3 wks. at C. H. (36839)	Bandage to abdomen 2 wks. spica 1 wk. then brought to C. H. with callus	Traction 5 days—X-ray no change	None	Yes—with plates show a slight lateral bowing—otherwise normal. No shortening.	An excellent case study.
16.	S. S. 9-2	154586	4 yrs.	M	Easy	Upper end lt. Antero Lat. Bowing Lt. trochanter and bene Nealton's line.	4 yrs. Came because of stiff hip, stiff knee gait.	4 yrs.	None	Massage exercise	Atrophy of leg. Abnormal gait.	No—did not return to Clinic	Born at Maternity Hospital.
17.	D. G. (Less than 2 mos. premature)	154057	16 mos.	M	Normal	Mid and upper 1/3. Marked lat. bowing.	16 mos.	16 mos.	None	Physiotherapy	None	One month. Progressively well.	
18.	E. B. 5	162625	9 mos.	M	Easy Vertex	Shortened end lt. femur at mid and upper 1/3 with lat. angulation	9 mos.	7 mos.	None	2 leg lengthenings with no bony union. X-ray pseudoarthrosis after operation.	Malunion	Yes—bad result. No bony union.	Child cried considerably first 3 mos. of life. X-ray suggests birth injury.
19.	W. F. 6-3	172441	16 mos.	M	—	Rt. midshaft with bowing laterally	Birth	Birth	Plaster spica 1 wk. then bandaged 2 wks.	Thomas heel rt.	Shortening of fractured leg		
20.	R. G. 10-8	131849	3 mos.	M	Difficult Breech	Low rt. femur with post. angulation Neck lt. femur with coxa vara Humerus mid 1/3	3 mos., humerus diagnosed at birth	3 mos.	Seen by attending physician but no treatment	Physiotherapy	Fractured humerus. Lt. coxa vara. Rt. post. bowing	Child lost to Clinic.	Attending physician diagnosed fractured humerus but not fractured femora. Advised no treatment.

BIRTH FRACTURES OF LOWER LEGS

	Name and Birth Wt.	Hosp. No.	Age	Sex	Labor and Type of Delivery	Location and Type	Time of Recognition	First X-Rays	Early Treatment	Late Treatment	Complications	Follow-Up	Remarks
1.	D. C. 8-10	137943	8 mos.	M	Easy Vertex	Lt. tibia mid 1/3. Lt. fibula lower end.	Symptoms noted 4 mos.	8 mos. (23401)	Immobilization failed to give union	Osteoperiosteal graft	No union	Patient last seen at 2 yrs. No union.	
2.	W. A. M. 9	163822	14 mos.	F	Difficult Breech	Rt. tibia and fibula	Birth	? 1 wk. at G. H. at 14 mos.	Not known. Baby said to have "healed" when mother was discharged from hospital.	Osteoclasis of left femur at 14 mos.		O. K. 3 mos. after osteoclasis	
3.	D. D. 3	184503	1 yr. 3 mos.	M	—	Rt. tibia and fibula	Birth	13 mos.	None	Osteotomy	Slow but definite union	Firm union not present at 5 mos.	
4.	A. M. S.	146942	3 wks.	F	Difficult Breech	Lt. tibia midshaft. Moderate overriding posterior angulation.	1 mo.	1 mo.	None	Much callus at 1 mo. No treatment felt indicated.	Hydrocephalus. Fracture right clavicle.	Child developed hydrocephalus	Neither clavicle nor fracture of lower leg noticed by attending physician

BIRTH FRACTURES OF THE HUMERUS

(Continued)

	Name and Birth Wt.	Hosp. No.	Age	Sex	Labor and Type of Delivery	Location and Type	Time of Recognition	First X-Rays	Early Treatment	Late Treatment	Complications	Follow-Up	Remarks
9.	K. G.	157383	2 wks.	M	Difficult Breech	Greenstick midshaft lt.	Birth	2 wks.	Plaster 2 wks., then swathe	Physiotherapy. Cockup splint to wrist and hand—at 4 wks. of age.	Radial nerve paralysis	Yes	This child diagnosed epilepsy at 2½
10.	C. C.	78093	1 day	M	Easy Breech	Lt. humerus midshaft Transverse	Birth	1 day	Swathe splint to support wrists		Radial nerve paralysis slight, O. K. at 1 mo.	Yes. Good result.	
11.	B. C.	80058	1 day	M	Normal Breech	Lt. midshaft mid and lower 1/3	Birth	1 day	Coaptation splints and swathe	None	Slight radial nerve involvement. Cleared O. K. with support to wrist.	Good result. Discharged at 1 mo.	
12.	B. L.	84269	13 days	F	Difficult Breech	Mid and lower 1/3 rt. outward angulation	3rd day	3 days at another hospital. Plates showed no fracture.	Swathe and sling	Not known	Not known	None	Noted by mother 3rd day.
13.	J. M.	88030	1 wk.	M	—	Transverse midshaft. Slight inward displacement.	1 wk.	1 wk.	Swathe 16 days	None	None	Yes	Good result.
14.	M. S. 8-15	102885	3 mos.	F	Difficult Instruments	Ununited fracture right humerus. Greenstick midshaft.	3 mos.	3 mos.	Swathe 1 mo., callus	Nerve suture and tenotomy of pectoralis	Whole upper arm type. Birth injury.	Yes. Final result not known. Child improved.	
15.	A. S.	148304	1 day	M	Normal foot present	Fract. rt. humerus. Lower 1/3 angulation and slight overlapping.	Birth	1 day	Fig. 8 swathe for arm	Fragments slipped. Skin traction 1 lb. wt.	Lost 8 oz. while on frame 1 wk.	Yes. Good result.	
16.	L. S.	107530	16 days	F	Del. by midwife	Complete transverse midshaft sl. overriding	Birth	16 days		None	None	One visit. O. K. then.	
17.	M. L.	134450	2 wks.	F	Normal Low forceps	Transverse midshaft lt.	Birth	2 wks.	Cardboard splints	Airplane splints rt. Coaptation splint lt.	Brachial palsy rt.	Yes. O. K. 1 mo.	
18.	J. H. 6-¾	112897	19 days	M	Difficult Breech	Lower end lt.	16 days	16 days here. X-rays by attend. phys. said to show dislocation.	None except manip. in attempt to reduce disiocation, arm bandage to side	Put up in extension 6 days, then exercise	None	Perfect result	Fracture not diagnosed by attend. phys. Took 2 x-rays and diagnosed dislo. Made 2 attempts to reduce "dislocation".
19.	J. B. 10	178029	8 mos.	M	Difficult Breech	Lt.	Birth	—	—	—	None	None	
20.	A. G. 8-½	155875	3 days	F	Normal Vertex Delivery of shoulders	Midshaft Greenstick Lt.	Birth	3 days	Splints ? type	None. Alignment 1 mo. perfect.	None	Good position 1 mo.	
21.	R. G. 10½	131849	3 mos.	M	Difficult Breech Forceps	Rt. mid 1/3 post. angulation	Birth	3 mos.	None	Orth. physiotherapy	Fracture lower and rt. femur. Neck lt. femur.	Yes, hamstrings stretched. ? result.	
22.	A. R.	90369	1 mo.	F	Difficult Low forceps	Lt. mid 1/3 sl. outward bowing	Birth	1 mo.	A. P. splints for 2 wks.	Lt. plaster for 2 wks. because fracture line still visible.	None	Yes	
23.	T. B. 10-¾	95538	4 mos.	M	Difficult Version	Mid 1/3 lt. lat. angulation	Birth	9 mos.	Board splints by attend. physician 4 wks. Deformity noticed on removal of splints.	None	Obs. paralysis upper arm, ? spinal cord injury	None	
24.	J. M. 8	98441	4 days	F	Normal Vertex	Rt. transverse midshaft medial displacement	Birth	4 days	Axillary pad with sling then swathe after 1 day	Cockup splint at 9 days because of wrist drop.	Radial palsy slight.	Yes. Good result.	
25.	J. H. 7	100845	1 wk.	M	Difficult Vertex delivery	Left supracondylar	4 days	11 days	Swathe to bind arm to body in flexion	Posterior gutter splint to hold arm at rt. angles. Baking. Massage.	Lt. wrist drop. Lt. enopthalmus.	Yes. Excellent result at 3 mos. Limitation of extension up to 1 mo.	
26.	J. F. 10	187407	11 hrs.	M	Difficult Vertex delivery	Lt. oblique midshaft	Birth	1 day	Swathe binding arm to chest for 1 month	Swathe binding arm to chest for 1 month	None except for slight maceration of axilla	Excellent result 1 mo.	
27.	E. M. 10-12	187551	3 wks.	M	Normal Multip. Vertex	Lt. oblique midshaft	Birth	1-2 days	Throat stick splints unsatisfactory	5 days change to swathe (ace) axillary pad. Then adhesive used.	Slight wrist drop which cleared with stretching and massage. Large callus.	Excellent result 1 mo.	Baby was given Viosterol, gtt. V daily during treatment of fracture. Large amount of callus formed. No evidence of injury.
28.	R. B. 7-8	106261	5 days	M	Difficult Difficult	Rt. complete transverse midshaft slight overriding	Birth	2 days	"Setting" by attending physician unsatisfactory	Arm wrapped and put in sling	Slight ? evidence of nerve injury for 2 wks. then disappeared	Solid. Good result in 3 wks.	
29.	R. B. 9-3	158817	15 days	M	Long difficult ? difficult	Rt. oblique midshaft	15 days	15 days (31365)	Arm bound to side with swathe	Physiotherapy because of ? Nerve injury	Nerve injury cleared after 1 mo. of muscle training	Yes. Excellent result.	Doctor did not recognize nerve injury or fracture. Child sent in because of sprain.
30.	W. B. 9-4	160419	2 wks.	M	Normal ? Normal	Rt. midshaft	Birth	1 day at Maternity Hospital	Reduction at 1 day with double moulded plaster splint with immobilization to trunk	2 wks. dorsiflexors weak—cockup splint. For 3 days then removed.	? slight nerve injury	Yes. Excellent result.	
31.	B. S. 7-½	163823	2 wks.	F	Difficult Instrumental version	Rt. oblique mid 1/3	Birth	17 days	None	Cockup splints 2 wks.	Radial nerve irritation	Yes. Solid. No nerve paralysis at 3 wks., 5 wks. after injury	Good picture.
32.	R. C. 7-6	184853	2 wks.	M	Difficult Breech	Rt. midshaft	Birth	2 wks.	Throat stick splints with sling	Same position O. K.	None	Yes. Solid with callus 24 days after injury	
33.	J. D.	191889	6 mos.	F	— —	Midshaft rt. humerus	Birth	? at C. H. at 6 mos.	Splinting by attending physician 6 wks.	Physiotherapy at 6 mos. for brachial palsy	Slight brachial plexus disturbance, also ? radial	Yes. Cleared with physiotherapy	
34.	J. C. 10	173597	38 days	M	Easy Normal vertex	Lt. spiral fracture. Midshaft humerus. No union.	38 days	38 days	None	Immobilization in abduction in plaster.	None	Yes. Firm callus 2 wks.	Good result.
35.	B. D. 8	149853	8 wks.	F	Difficult Breech	Rt. midshaft. Good position.	Birth	By attending physician	Splints bandage	None	None	When seen here at 8 wks. position good	
36.	J. D. 10	146535	10 days	M	Difficult Breech	Lt. midshaft 1 cm. overriding	Birth	10 days	None	None	Fractured skull, transection of cord, flaccid paralysis of lower extremities	Died after 12 days in hospital Bronchopneumonia	
37.	S. K.	115460	2½ mos.	F	Difficult Breech Version with breech	Rt. midshaft humerus	Birth	2½ mos.	None	Muscle training	Bad brachial palsy	Improvement with muscle training	Child lost to Clinic.

FRACTURE OF THE CLAVICLE

	Name and Birth Wt.	Hosp. No.	Age at Entrance	Sex	Labor and Type of Delivery	Location and Type	Time of Recognition	First X-Rays and No.	Early Treatment	Late Treatment	Complications	Follow-Up	Remarks
1.	B. R. 7-1	131705	2½ wks.	F	Normal low forceps Vertex—F. T.	Middle and outer 1/3 Rt.	At discharge from Maternity Hospital—13 days	2½ wks. showed dense callus position O. K. (20318)	None	None	None		Never seen by surgeons.
2.	S. G. 10	131598	4 wks.	M	Type instrumental F. T.	Midportion lt.	8-2-29 2 days Maternity Hospital	8-2-29 (19988)	None	None	None	10-9-29—X-ray and good callus and position	
3.	H. M. 9	122741	2 wks.	M	Instrumental used to shorten 2nd stage Vertex F. T.	Rt. Clav.	2 wks.	2 wks. 11-26-28 (17139)	None	None	None		Fracture picked up on x-ray for "enlarged thymus". Symptoms—cry on inspiration. Cyanotic attacks. Had enlarged thymus.
4.	P. 8	122066	2 wks.	M	Difficult F. T.?	Midportion Lt.	c. f. delivery	11-1-28 2 wks. (16877) good callus and position	None	None	None	None	Never seen by surgeons.
5.	J. C. 12-3	118831	3 wks.	M	Difficult type	Distal 1/3 without apposition of fragments lt.	18 days	2 wks. at B. C. H.	None	None	None. Free motion of arm and shoulder.	1 month after entry showed firm union by P. E.	No x-rays at this Hospital. Not picked up by attending physician.
6.	E. D. 8-6	118386	3 wks.	M	Primip. and forceps Vertex	Rt. midshaft. Downward displacement of outer fragments sl. overriding.	At discharge from Maternity Hospital	3 wks. (13691)	None	None	None	None	
7.	J. M. 10	116413	2 wks.	F	Difficult Breech extraction under ether	Middle and outer 1/3. Left slight downward angulation.	1 day	2 wks.	None	None	None	None	
8.	J.	110929	1 mo.	M	Difficult Diff. breech Maternity Hospital	Left	At delivery	None	None	None	None. Seen by Neurologist. No paralysis.	None	Fracture palpated as well healed.
9.	A. T. 8-½	107323	1 mo.	M	Normal Normal	Rt. midshaft	Age 3 wks. Lump noted by parents.	1 mo. (9834)	None	None	None	None	Not diagnosed by attending physician.
10.	J. M. O. 8-14	157765	8 days	F	Long High forceps	Left midportion	7 days by parents	8 days (sheet not on record)	Chest swathe 8-15 days	None	None	None	Not diagnosed by attending physician.
11.	B. B. 13	106200	16 days	F	Long Low forceps very difficult	Rt. mid and outer 1/3	16 days by x-ray	16 days (9514) dense callus	None	None	Brachial palsy obs. Rt. noted at 2 days.	None	Diagnosis made by x-ray. Attending physician and O. P. D. physician did not pick up fracture.
12.	A. L.	105737	2 wks.	M	Normal Normal	Left midshaft. Slight downward angulation of outer fragment.	Birth by parents	2 wks. dense callus	None	None	Slight deformity at 4 wks. of age		Diagnosis of fracture made in S. O. P. D. not by attend. physician.
13.	C. T.	104898	6 wks.	F	Difficult with high forceps high forceps	Mid and out. 1/3. Good position.	6 wks.	6 wks. (9160)	None	Muscle training	Hematoma of lt. sternomastoid with torticollis Erb's paralysis	By muscle training	Paralysis but not fracture diagnosed by attending physician.
14.	M. H. 8-10	102203	2-½ mo.	F	Normal Normal Multip.	Rt. midshaft. No displacement	12 days on discharge from Maternity Hospital	2-½ mos.	None	None	None	None	
15.	C. T. 7-10	101634	1 mo.	M	Multip. Normal Normal	Mid. rt. slight downward displacement of outer fragment	34 days at C. H.	34 days	None	None	None	Good union at time seen at 1 mo. 2 and 3 mos.	Diagnosis not made at Maternity Hospital discharge. Made at C. H. Clinic.
16.	M. F.	100011	21 days	F	Normal Instrumental	Mid. 1/3 lt.	20 days by parents	None	None	None	None	None	Recognized by parents.
17.	R. G. 9-¼	99960	11 days	M	Normal Normal	Midshaft Much callus lt.	4 days by parents	11 days	None	None	None	Yes. O. K. at 3 mos.	Recognized by grandmother.
18.	J. F. 6-½	96043	3 mos.	M	Normal Normal	Mid. 1/3. No displacement rt.	Shortly after birth by mother	3 mos.	None	None	None	None	Recognized by mother.
19.	S. H. 7-9	90919	3 wks.	F	Normal Primip. Normal	Rt. mid. No displacement.	On discharge from Maternity Hospital	3 wks.	None	None	None	Yes—1st plate—Fracture. 12 days after plates: good union without displacement.	
20.	D. L. 8-3	90913	3 wks.	M	Normal Multip. Normal	Mid. and outer 1/3 alignment O. K.	On discharge from Maternity Hospital	3 wks.	None	None	None	Yes—as above.	
21.	R. R. 9-½	86781	1 mo.	M	Difficult high forceps	Mid. rt.	At birth	None	None	Muscle Training	1. Obs. paralysis—upper arm type. 2. Torticollis with hematoma.	Came to Muscle Training Clinic 2x, then did not come back further.	Referred by attending physician.
22.	J. S. 9-14	90175	3 wks.	M	Normal Normal	Midshaft lt. Outer frag. overriding the inner good callus.	At Maternity Hospital	4 wks.	None	None	None	Yes—Union O. K.	
23.	J. F. J. 8-4	84203	5 wks.	M	8 hours Normal	Midshaft slight upward angulation—lt.	At 2 wks. by parents	5 wks.	None	None	None	No	Recognized by parents.
24.	M. C.	78825	2-½ mos.	F	Very difficult Difficult	Midshaft rt.	At birth	None	None	Arm tied up for 10 days at age of 7 wks.	Arm weak temporarily but function returned	No	Recognized by attending physician.
25.	B. F. 8-12	178070	2 wks.	F	Multip. Breech	Rt. mid and outer 1/3. Moderate overriding. Callus present.	At birth Maternity Hospital	2 wks.	Fig. 8 bandage. Clavicular above pillow at 2 wks.	None	None	No	Recognized at Maternity Hospital.
26.	E. C.	173994	2 wks.	F		Lt. outer and mid 1/3	By parents 10 days	2 wks.	None	None	None	Yes O. K. at 5 wks.	Recognized by parents.
27.	V. M. 8-12	168139	1 mo.	F	Primip. Delivery low forceps	At outer 1/3 and inner 2/3 lt. good position	At Maternity Hospital discharge	11 days	None	None	None	Yes at 1 year O. K.	
28.	M. B. 7	164987	1 mo.	F	Normal	Lt. midshaft	At 2 wks. by parents	1 mo.	None	None	None	None	Recognized by parents.
29.	E. C. 8-15	164055	5 wks.	M	Difficult Instrumental	Lt. midshaft	Noted by mother	5 wks.	None	Muscle exercises for torticollis	Rt. torticollis with ? hematoma of sternomastoid	Yes—improved	Recognized by parents.
				F	Normal	Greenstick mid 1/3 lt.	At birth at Maternity	1 day	None	None	None	Yes—O. K. at 1 year	

	13				Vertex Difficult									
32.	S. C. 7-2	159391	2 wks.	F	Difficult Breech Primiparous	Mid inner 1/3 left	At birth Maternity Hospital	2 wks.	Yes--splinted	None	None	Yes—O. K. at 2 mos.		
33.	J. W. 7-½	159318	4 mos.	M	Difficult Breech Instrumental	Rt. midportion	2-3 weeks by parents	4 mos.	None	Physiotherapy	Rt. torticollis with hematoma of sternomastoid	Yes—no abnormality at 2½ mos. Followed to 8 mos.—O. K. (after entry).	Recognized by parents.	
34.	C. T. 7	159109	2 mos.	F	Normal Normal	Lt. mid and outer 1/3	1 mo.	2 mos.	None	None	None	Yes—until 7 mos.	Recognized by parents.	
35.	E. D. 10	159344	11 days	F	Difficult delivery of shoulders. Difficult shoulders extracted normally.	Lt. midshaft	At birth	11 days	Fig. 8 bandage	None	None	Yes. O. K. at 1 mo. after entry.		
36.	M. O. 7-9½	157282	6 wks.	F	At C. H. No history.	Rt. mid 1/3	2 wks. by parents	6 wks.	None	None	None	Yes. O. K. at 2-3 mos. after entry.	Recognized by parents. Diagnosis of dislocation by attending phys.	
37.	E. C. 6-6	150899	5 wks.	M	Difficult Breech	Lt. mid 1/3 with considerable overriding	4 wks. by mother	5 wks.	None	Fig. 8 bandage 1 wk.	None	Yes. O. K. 1 mo.	Recognized by parents.	
38.	J. B. 7-6	149140	1 mo.	F	Difficult high forceps	Lt. mid 1/3	At birth	1 wk. private 1 mo. here	None	Splint for paralysis. Phys. Th.	Obs. paralysis. Upper arm type.	Yes. 2 yrs. with nearly complete cure, still followed.	Diagnosed by x-ray.	
39.	A. S. 9	146942	3 wks.	F	Difficult. Difficult Breech.	Rt. mid and outer 1/3	3 wks.	3 wks.	None	Muscle Tr. for Erb's paralysis.	Obs. paralysis. Upper arm type.	Pt. developed hydrocephalus. Improved with M. T. at 3 mos., then did not return.	Not picked up on examination in this Clinic.	
40.	A. B.	145267	5 wks.	M	— —	Rt. inner 1/3	12 days (about)	5 wks.	None	None	None	None	Noted by parents.	
41.	B. M. 8-3¼	143984	2½ wks.	F	Difficult Instrumental	Rt. mid 1/3	At birth	10 days at Maternity Hospital	None	None	None	None	Recognized at birth at Maternity Hospital.	
42.	R. H. 9-11	143867	2 wks.	M	Difficult Difficult	Lt. outer 1/3. No displacement.	Birth by attending physician	2 wks.	None	Muscle training	Brachial palsy whole arm (mostly upper)	Yes. See M. T. record.	Recognized at birth by attending physician.	
43.	E. G. 7-13½	142504	4 wks.	M	Difficult	Lt. mid 1/3	Birth by attending physician	None	None	None	None	None	Recognized at birth at Maternity Hosp.	
44.	G. E.	141711	2 wks.	F	Difficult forceps	Lt. mid 1/3	Discharge Maternity Hospital	2 wks.	None	None	None	Patient did not return	Recognized at birth at Maternity Hosp.	
45.	J. K. 8-12½	136403	6 wks.	M	Difficult Breech	Rt. outer 1/3	Discharge Maternity Hospital	None	None	None	None	Yes. O. K. at 2 mos.	Not seen by surgeons.	
46.	L. 7-10	135465	2 wks.	F	Difficult	Complete downward angulation of outer fragment. Lt. mid and outer 1/3.	7 days at Maternity Hospital	(21891)	Fig. 8 splint	None		Yes. Good result.	Noted by mother at 7 days while in Maternity Hosp.	
47.	E. R. 13-¼	136223	5 wks.	M	Difficult Difficult	Rt. mid 1/3	At birth	By attending physician at 3 days	Arm tied up 1 wk.	M. T. right brachial palsy	Yes. Lt. brachial palsy ? sl. spastic quadriplegia.	Yes. By M. T.	Recognized by attending physician.	
48.	D. C. 8-2	135077	3 wks.	F	Normal Difficult Multip.	Lt. mid 1/3	At Maternity Hospital discharge	3 wks.	None	None	None	Yes	Diagnosed at discharge from Maternity Hosp.	
49.	A. S.	148304	1 day	M	Difficult Foot presentation with breech delivery	Rt. outer 1/3 downward displacement of outer fragment	At birth by attending physician	1 day	Skein Fig. 8 arm to clavicle ace bandage to immob. rt. arm to chest	See fract. of humerus	Fractured rt. humerus	Yes. Good result.	Recognized by attending physician at birth.	
50.	D. M. 7-2½	178624	2 wks.	M	Normal Low forceps	Lt. mid 1/3	Birth	2 wks.	Yarn truss to clavicle	None	None	None. Good result.		
51.	M. H. 8-15	194609	1 mo.	F	Primipara	Rt. mid 1/3	2 wks.	1 mo.	Yarn truss to clavicle	None	Rt. facial palsy	Facial palsy clearing		

BIRTH FRACTURES OF THE HUMERUS

	Name and Birth Wt.	Hosp. No.	Age	Sex	Labor and Type of Delivery	Location and Type	Time of Recognition	First X-Rays	Early Treatment	Late Treatment	Complications	Follow-Up	Remarks
1.	E. M. 8-15	170641	3 mos.	M	Difficult. Delivery vertex.	1. 2 cm. below upper end. 2. Transcondylar.	3 mos.	3 mos.	None	Post. plaster splint. Obst. splint.	Lt. upper arm obst. palsy at birth	10 mos. later child can raise arm to level of ear.	Fracture not noted until during a return to Muscle Clinic, when arm was found swollen and tender. X-ray showed fracture.
2.	R. L.	119304	6 days	M	Instrumental	Midshaft lt. humerus	Birth	6 days	Plaster for 10 days	None	None	None	
3.	P. S.	112076	2 wks.	M	Breech	Midshaft lt. humerus	Birth	2 wks.	Put up by attending physician. Slight wrist drop present. Bandage tight.	Put up with axillary pad and swathe 4 days and sling for 12 days	Wrist drop from too tight bandaging	Wrist drop cleared, good result.	
4.	L. F.	112490	19 days	F	Breech after version	Rt. midshaft humerus	Birth Maternity Hospital	(11698)	Velpeau with cardboard splint. Callus good but refractured.	Pistol holster sling—11 days	Slight excoriation under skin	Good result	
5.	C. P.	110368	2 days	M	Easy. Vertex, 1 arm over head.	Oblique lt. midshaft. Position good.	Birth	2 days	Coaptation cravat sling and swathe		Child developed mastitis. Swathe had to be removed.	Yes. X-ray 1 month shows firm union in good position.	
6.	N. S. 9-½	154603	3 mos.	M	Normal	Rt. midshaft	Birth	3 mos.	Not known	None	Brachial palsy on lt.	None	Seen only once in Clinic.
7.	A. G. 8-½	155875	3 days	F	Normal. Difficult delivery of shoulders.	Green stick lt. midshaft humerus	Birth	3 days	Put up in splints, exact nature not mentioned	None	None. Good result. No paralysis.	For 1 month	
8.	M. S.	123106	5 hrs.	F	Difficult Version. Rt. arm extended over head.	Transverse rt. midshaft, sl. lateral angulation	Birth	5 hrs.	Arm bound to side with swathe			Union firm with callus at 3 wks.	

(OVER)

sidered responsible. Unrecognized and untreated fractures in this age group heal by the process of natural repair, but always with more deformity, sometimes permanent, than when adequate treatment is promptly applied. Repair of bone in this age group is so rapid that after 5 days of age it is impossible to reduce these fractures manually. The necessity of early diagnosis for effective treatment is of the utmost importance.

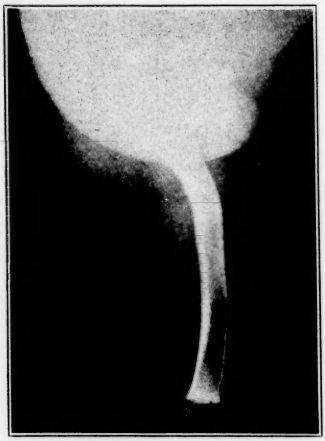

ILLUSTRATION NO. 4.
Case No. 129415—Healed Fractured Femur.
Lateral view 10½ months after fracture. No shortening. Natural repair aided by fixation in overhead traction.

Adequate treatment of fractures in the newborn consists in: (1) early diagnosis; (2) prompt realignment of the fragments; and (3) proper fixation until union has taken place. The early recognition of these fractures is essential if anatomic alignment is the aim of treatment. Many argue that nature repairs these fractures even if untreated (Pritchard and Smith[8]) but, excepting cases of clavicle fracture, we report that in 27 cases in which the treatment was considered inadequate 16, or 60 per cent, resulted in some deformity of bone. (See illustrations 1 and 2.) The reduction of these fractures does not involve the complicated manipulative procedures (no spasm) required in older children and in adults, but merely an accurate realignment of the fragments. When this is properly accomplished, useful and appropriate methods of fixation are at our disposal. A description of these types of fixation is presented in the accompanying table and illustrations.

These recommended methods for fixation have produced good results. Although many methods have been described in the literature,[7, 9, 10, 11] these presented here are preferred because of their simplicity, uniformity, and ease of appli-

TABLE 1

Bone	Type of Fixation	Period of Fixation
Clavicle	Figure of 8 with skein yarn	3-4 weeks
Humerus	Axillary pad of wadding; coaptation splint; internal angular splint; or moulded plaster. Swathes.	4-5 weeks
Femur	Overhead skin traction of both legs on Bradford Frame with 2-3 lbs. weight.	4-5 weeks
Tibia, or	Plaster, toes to groin.	4-5 weeks
Tibia and Fibula	" " " "	4-5 weeks

cation. A brief summary of results obtained in this series of 115 cases follows and confirms our belief in the adequacy of these methods.

ILLUSTRATION NO. 4a.
Case No. 129415—Healed Fractured Femur.
Ten and one-half months after fracture. No shortening. A. P. view of case shown in Illus. 3 and 4.

Many cases of clavicle fracture heal well even though untreated and are frequently recorded when the parent discovers a callus enlargement after union has taken place. However, early diagnosis and application of a skein of yarn

	Recognized by parents.
	Recognized by parents.
	Recognized by parents. Diagnosis of dislocation by attending phys.
	Recognized by parents.
	Diagnosed by x-ray.
	Not picked up on examination in this Clinic.
	Noted by parents.
	Recognized at birth at Maternity Hospital.
	Recognized at birth by attending physician.
	Recognized at birth at Maternity Hosp.
	Recognized at birth at Maternity Hosp.
	Not seen by surgeons.
	Noted by mother at 7 days while in Maternity Hosp.
	Recognized by attending physician.
	Diagnosed at discharge from Maternity Hosp.
	Recognized by attending physician at birth.

Remarks

	Fracture not noted until during a return to Muscle Clinic, when arm was found swollen and tender. X-ray showed fracture.
	Seen only once in Clinic.

(OVER)

in a figure of 8 are recommended (fig. 1). In 20 cases of humerus fracture treated with coaptation splint, internal angular splint, or moulded plaster, axillary pad and swathe, results were good, whereas in 50 per cent of those treated by other methods the results were unsatisfactory. In every instance of femur fracture treated with overhead skin traction on a Bradford frame (figs. 4 and 5) the end-result was satisfactory, whereas in 100 per cent of those treated with adhesive strapping of the leg to the abdomen, unsatisfactory results were obtained necessitating a change in the treatment after a short trial. In only one case in this group was a fracture of the lower leg reported early enough to have appropriate reduction and fixation applied, deformity being already present in 75 per cent at the time of admission to the hospital. Another case resulted in nonunion after a bone graft.

An important adjunct to the method of fixation of these fractures in the newborn is the hygiene and care of the baby's skin. Adequate padding and skin hygiene is essential to avoid the appearance of pressure sores, and other complications. Especially is it of the utmost importance in the fixation of fractures of the femur to allow for careful hygiene of the skin of the buttocks. The authors believe that fixation of these fractures by overhead adhesive traction is the one method most adaptable for convenient diaper hygiene.

We cannot subscribe to the old teaching that birth fractures always heal spontaneously and

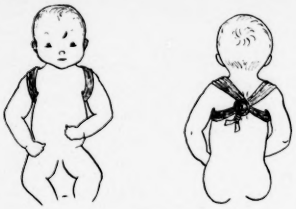

FIGURE 1.
Figure of 8 principle using a skein of white yarn, for fracture of clavicle.

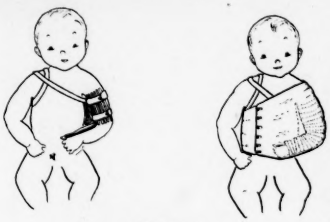

FIGURE 2.
Axillary pad of sheet wadding, coaptation splints, and internal angular splint or moulded plaster, with swathe, for fracture of the humerus.

FIGURE 3.
Plaster of Paris, toes to groin with knee flexed for fracture of the tibia and fibula.

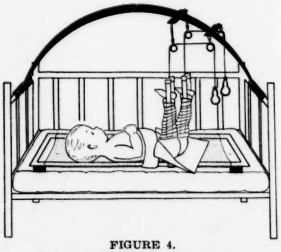

FIGURE 4.
Skin traction with adhesive and 2 or 3 lbs. of weight with vertical pull, on rainbow frame for hospital treatment of fracture of the femur. Note both legs.

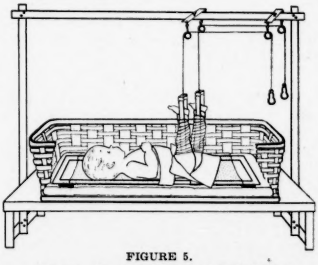

FIGURE 5.
Same as fig. 4, only using laundry basket and homemade wooden overhead frame.

satisfactorily regardless of any treatment instituted. As recently as 1934 Pritchard and Smith[8] propounded this thesis before the Royal Society of Medicine and were severely criticized in the discussion of their paper. It is the aim of our paper to emphasize that improved meth-

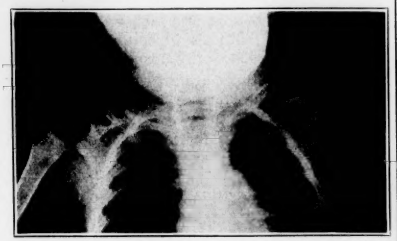

ILLUSTRATION NO. 5.
X-Ray No. 37544—Cleidocranial Dysostosis.
Six months of age. Thought to have been an ununited birth fracture of the clavicle.

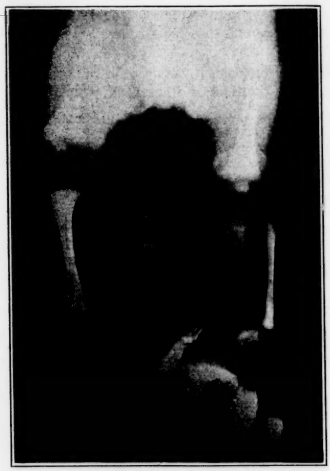

ILLUSTRATION NO. 6.
X-Ray No. 27962—Dysostosis of Tibia.
This may have two fragments (distal as well as proximal).

ods of treatment of birth fractures certainly are yielding better results. For early diagnosis, prompt and appropriate fixation in good alignment are essential to the attainment of good results.

The correct differential diagnosis of birth fractures is also important. Apparently cases of cleidocranial dysostosis have been wrongly considered as birth fractures. Other cases of tibial dysostosis may be found which have been classified incorrectly as fractures in the newborn. Fitchet[12] has described most completely the anomaly of cleidocranial dysostosis, which is actually an intrauterine developmental anomaly of unique nature. The author found one old case indexed in the files of the Children's Hospital as intrauterine fracture of the leg which actually was a dysostosis of the tibia. Cases of osteogenesis imperfecta and of fragilitas ossium have also at times been classified as birth fractures, particularly if the first fracture occurred at the time of delivery. This also is a develop-

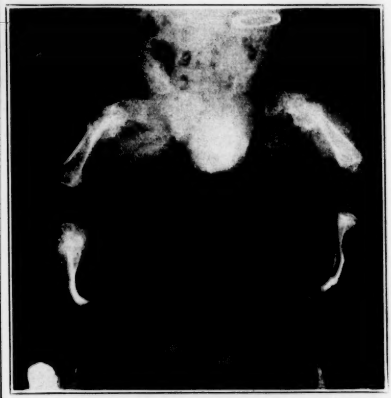

ILLUSTRATION NO. 7.
X-Ray No. 34332—Osteogenesis Imperfecta.

mental anomaly of utterly dissimilar nature. Since the pathology originates during intrauterine development, these three conditions should never be classified as true fractures in the newborn.

Excluding these intrauterine pathologic conditions, true intrauterine fracture may occur rarely, as described by Smith.[13] In 35 cases of 43 collected by him the pregnant mother had experienced severe trauma to the abdomen (gunshot, sickle, pitchfork, heavy falls and body blows). In our series, however, not one proved instance of true intrauterine fracture was discovered.

It is perhaps preferable to record the complications of birth fracture in table 2.

An important consideration in attempting to reduce the incidence of birth fractures is the control of body weight and size of the fetus. From the 90 histories in which the birth weight was known, reviewed in this series, a table has been made of the average birth weights in each bone fracture (table 3).

It is evident that, in every bone fractured, the average weight was above the normal. Since the fetal weight, according to Abels and others[14, 15, 16] may be influenced by regulation of the maternal diet, it would appear from table 3 that more careful attention might be paid to this item during prenatal care. So controversial, however, is this subject, that space does not permit its discussion.

TABLE 2

No. of Cases	Birth Fracture	Complications from Birth Trauma and Treatment	
53	Clavicle	Brachial palsy	7 (one temp.)
		Torticollis—sternomastoid rup.	4
		Facial palsy	2
		Fractured humerus	1
38	Humerus	Brachial palsy	6
		Musculo-spiral palsy	11 (all temp.)
		Fractured skull with transection of cord	1
		Fractured femur	1
		Fractured clavicle	1
20	Femur	Nonunion—(after operative intervention to correct deformity)	1
		Fractured humerus	1
3	Tibia and Fibula	Fracture of clavicle	1
1	Tibia	Nonunion—(after operative bone graft for nonunion)	1

TABLE 3

Bone	Wt. Recorded in	Average
Clavicle	44 cases	9 lbs.
Humerus	24 cases	8 lbs., 14 ozs.
Femur	20 cases	8 lbs.
Tibia and Fibula	2 cases	8 lbs., 13 ozs.

TABLE 4

Bone	Type of Delivery Recorded in	Percentage of Abnorm. Deliveries
Clavicle	44 cases	67%
Humerus	31 cases	75%
Femur	20 cases	85%
Tibia and Fibula	3 cases	67%

A second important factor in reducing the incidence of birth fractures is the type of presentation at the time of labor. Table 4, made from 98 cases in which the type of delivery was recorded, illustrates clearly that the majority of these cases of birth fracture required manipulative procedures to produce a live baby.

CONCLUSIONS

1. The incidence of birth fractures to all fractures as seen in a review of 6,000 cases at the Children's Hospital, Boston, reveals 113:6000 or approximately 1:60.
2. Birth fractures are, in the large majority of cases, accidents occurring in difficult obstetric manipulations.
3. Birth fractures in this series occurred in most cases in large babies.
4. Not one instance of true intrauterine fracture was found in this review. The disease entities of osteogenesis imperfecta, fragilitas ossium and the dysostoses should not be considered as included in true birth fractures.
5. The pleas for more adequate treatment in fractures of the newborn consist in (1) early diagnosis; (2) early manipulative alignment of the fragments involved; and (3) proper fixation.
6. An outline for methods of fixation with illustrations is presented.

NOTE: The authors wish to express their appreciation of the permission granted them by the Orthopedic Service of including in this series a few of its cases of unilateral bowleg.

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RECENT ADVANCES IN THE TREATMENT OF RECTAL DISEASES BY INJECTION METHODS IN AMBULATORY PATIENTS*

II. Pruritus Ani

BY NAAMAN STEINBERG, M.D.†

PRURITUS ani has probably been common to man throughout the ages. As early as 1688 it attracted medical comment and was considered a distinct entity. In 1694 Josephus Langonius[1] mentioned and described a severe perianal itch which he called a "pruritus". Thinking he observed a certain periodicity of the disease, he sought to explain it on the basis of the then current astrologic systems as being related to the solstice of the sun. From then on, it was mentioned only occasionally in medical writings and textbooks until the beginning of the twentieth century. In recent years an increasing interest in the subject has led to numerous studies, and a number of valuable therapeutic measures have been proposed.

Many are of the opinion that the condition is merely a symptom; others consider it a distinct clinical entity. Various classifications have been proposed. Gant,[2] for example, divides the etiology into several groups: 1. Constitutional diseases, such as diabetes, gout or hepatic disease. 2. Local perianal diseases, such as hemorrhoids, fissure, fistula, cryptitis or proctitis. 3. Parasitic, skin and fungus diseases, such as scabies, pediculi, eczema or epidermophytosis. 4. Reflex disorders caused by disease of the pelvic organs. 5. Idiopathic or "pruritus essentialis", as described by Tuttle. Many included in the last category would naturally be listed in one of the first four groups, if more concerning the nature of the disease were known.

Montague[3] presents two main groups: A. The direct type, where there is definite evidence of disease in the region of discomfort. B. The indirect type, where the itching is probably referred from some other region such as the stomach, the gallbladder or the appendix. His explanation of itching is analogous to that for referred pain from disease of a viscus.

Pruritus ani does not require definition. The itching is frequently exaggerated at night, resulting in insomnia and impairment of health. In addition to itching in the immediate anal region there is often extension to the scrotum and the perineum. The skin usually presents a whitish appearance and may be smooth and glistening. In some it is thrown into many deep folds radiating from the anal orifice. When the condition has been present for some time there is generally definite induration. Abrasions and excoriations may be present when there has been scratching and the skin is often red and denuded.

What are we to do for those patients in whom the process appears to be entirely local? The course of treatment, when the local itching is due to some general cause, such as diabetes, is obvious. But, even here, when the primary factor has been taken care of, the local itching may still persist. The same may be true when pruritus is related to such proximal conditions as hemorrhoids and tabs. The removal of the latter may not result in a cure, as is true with pruritus associated with a skin lesion elsewhere. Soon after itching occurs changes in the skin result probably because of the trauma of scratching, these factors keep the process going and a vicious circle is established.

Before the twentieth century, the treatment consisted in applying various lotions, ointments and powders locally. Thereafter the nervous innervation was attacked by surgery and by the subcutaneous injection of various solutions. Sir Charles Ball[4] in 1905 was the first to attempt a surgical method. Later, others modified the technic. The superficial nerve filaments in the affected area were severed by subcutaneous incision. Relief was accomplished. Regeneration, however, occurred in most instances with recurrence of itching in from 3 to 6 months.

Murray[5] attempted to show that pruritus was an infection caused by *Streptococcus fecalis* and he claimed good results by treating the patient with autogenous vaccines. Others could not confirm his claims.

Rolfe[6] used ionization, solutions of zinc or iodine being applied by special anal electrodes to the affected regions. Repeated treatments were necessary. The results were dubious and the relief of short duration at most.

Ultraviolet ray therapy and diathermy have also been used with little relief.

Kolde[7] advocated x-ray therapy and, surprisingly, claimed good results in women, but poor results in men. Pruitt[8] and Lockhart-Mummery[9] both report "15 per cent cures" by this method. A "15 per cent cure" should not be taken seriously unless properly controlled. If x-ray is to be used, care should be exercised in selecting the radiologist, for improper administration results in serious complications. In

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isolated instances one encounters excellent results with x-ray therapy.

Much more satisfaction has followed the injection of various solutions which act by injuring the nervous supply of the perianal skin. Stone[10, 11] in 1916 was the first to use 95 per cent ethyl alcohol in minim doses, injected just below the perianal skin about 5 millimeters apart throughout the affected area.

Buie[12] used 40 per cent ethyl alcohol. He felt that the patient should be hospitalized, and, if anal disease is present, such as hemorrhoids, fissure or tabs, it must be eliminated and the tissues completely healed before treatment. He injects up to 20 cc. of this solution on each side, subcutaneously, in the exact manner as for local anesthesia. The treatment requires constant observation during a period averaging 6 weeks. Great care should be taken following the injection to avoid inflammatory reaction, abscess formation and sloughing. He states, "Although sloughing cannot be avoided in half of the cases in which the injection is given, it is possible to keep it from going too far by careful and diligent measures in all."

Hanes[13] experimented with dilute hydrochloric acid and found that a 1:3000 dilution could be injected with safety. Because of the pain accompanying this injection, the use of this solution must be preceded by the injection of at least 25 cc. of 0.5 per cent novocaine. Amounts varying from 2 to 6 ounces are used, depending upon the particular case, and treatments may be repeated in from 1 to 3 weeks.

The following medicinal solutions in oil have been used for subcutaneous injections: 1. Benacol (a mixture of equal parts of para-amino benzocaine benzoate and phenmethylol in 90 parts of rectified sweet almond oil) by Yeomans et al.[14] 2. Gabriel's[15] original A. B. A. solution (anesthesin 3 per cent, benzyl-alcohol 5 per cent and ether 10 per cent in sterilized oil). 3. Phenol in oil by Goldbacher[16]. 4. Anucaine (5 parts each of benzocaine and phenmethylol, 1 part of butyl amino-benzoate in one eighth part of basic procaine in sweet almond oil) by Gorsch[17]. 5. Gabriel's[18] modified solution (nupercaine base 0.5 per cent, benzyl-alcohol 10 per cent and phenol 1 per cent in 5 cc. of sterilized sweet almond oil).

Beginning in 1926 we attempted to treat this condition by the injection of large quantities of quinine and urea hydrochloride, 0.5 per cent, subcutaneously in amounts varying from 25 cc to 75 cc. Only a small percentage of cases obtained permanent relief, the greater number having a recurrence of symptoms in from 1 to 3 months. The use of A. B. A., benacol, and phenol in oil solution greatly improved our results. Following the use of these solutions, however, many patients experienced extreme discomfort in the injected area which lasted from several hours to days in some cases and often required sedatives and morphine.

Since Gabriel's modified formula contained the anesthetic, the alcoholic and the phenol treatments combined in one, it offered the most logical oil-soluble solution to be used in ambulatory cases. Anesthesia from nupercaine (percaine) in watery solution usually lasts from 4 to 6 hours; that from the base lasts even longer. The phenol is added to intensify the anesthetic power of the nupercaine, the alcohol to destroy the nerve filaments supplying the skin. All these medicaments in oil have a prolonged anesthetic effect which generally persists from 2 to 6 weeks. Due to the fact that there is no pain following the injection, no danger to the patient of the formation of large abscesses or great areas of sloughing and no pain to the injection itself except the first sensation of the prick of a needle, this treatment can be administered to the patient in the office. If itching recurs the patient returns for further treatments, fearing neither pain nor incapacitation.

In Frankfeldt's[19] series of 65 cases, based on treatment with this solution, he obtained cures in 40 per cent, marked improvement in 20 per cent and satisfactory results in 20 per cent of cases. The results in the remaining 20 per cent were unsatisfactory.

The present communication presents results obtained in 100 cases treated between 1933 and 1936. Each patient was followed for a period of at least 2 years. There were 59 males and 41 females, varying in age from 20 to 65 years. The greatest incidence (39 per cent) was in the fourth decade.

TECHNIC

The perianal skin is cleansed with tincture of green soap and painted with Scott's solution (mercurochrome crystals 2 parts, distilled water 35 parts, acetone 10 parts and alcohol 55 parts) or tincture of merthiolate (1:1000). One ampule of nupercaine-phenol-benzyl alcohol in oil, which has been slightly warmed to facilitate the flow of oil, is drawn into a 5 cc. sterile glass syringe through a large caliber needle. The needle is then changed to one of gauge No. 21 1¾ to 2 inches long. The perianal region is then divided into four quadrants, and usually either the left or right posterior quadrant is chosen first. The patient should lie in the Sims' position, on the side selected for the treatment. The needle is inserted subcutaneously about one half inch outside the affected area. *It must be freely movable at all times.* The solution is then injected in fan-shape manner until all the 5 cc. have been used. *The injected tissues are massaged gently for about 3 minutes* with a sterile piece of gauze, thus assuring an even distribution of the oily solution. The second

injection is given 2 days later in the opposite posterior quadrant, and, at 2 day intervals, the two anterior quadrants are treated in a like manner. *Great care should be taken not to inject the solution too superficially or intradermally, as sloughing will invariably follow.* The external sphincter muscle should not be injected, because if more than one quadrant of the muscle has been injected, the anal canal may become patulous and temporary loss of control take place.

After the injections have been completed the skin becomes less indurated, its color becomes more normal and the whiteness and excoriations disappear.

RESULTS

There were 24 patients who were entirely free of symptoms for a period of 2 years or longer after having received but one course of treatment. A second group, comprising 27 patients, showed recurrences at the end of from 10 months to 2 years; a second course (one to three treatments) resulted in no further recurrence during 2 years. A third group of 21 patients showed recurrences at the end of 6 months and in the course of 2 years a second recurrence, which finally responded to a third series of treatments. The fourth group of 16 patients recurred periodically every 3 to 4 months, but were regularly made comfortable after each series of treatments, consisting of from two to four injections. A group of 11 patients showed recurrences every 1 to 2 months, and this form of therapy was finally abandoned.

There is no question that the result of injection is prompt relief in almost all cases and this relief as indicated, varies from 1 month to more than 2 years. The particular result in any case is very probably merely an indication of the local conditions prevailing. Fifty-one per cent show results that may be called excellent and in 22 per cent the results were satisfactory. Even in the most severe cases, great relief was obtained for at least a month.

COMPLICATIONS

No serious complications have been observed after this form of treatment.[14, 15, 16, 17, 19, 20] In the present series of over 800 separate injections there were five superficial chemical abscesses of small size which were incised without any further anesthetic and healed without fistula formation. There were also eleven instances of superficial sloughing which caused slight discomfort for several days. Healing took place within 2 to 5 weeks. There were no systemic reactions.

SUMMARY AND CONCLUSION

Results with Gabriel's modified solution in the treatment of pruritus ani in 100 cases, followed over a period of 2 years or longer, are described. In 73 per cent the results were satisfactory, in 51 per cent they were excellent and in all instances at least temporary relief was obtained.

The advantages of this form of treatment are as follows: the high incidence of good results, the simplicity of the technic, its freedom from complications and after-pain and its ambulatory character, a great economic factor to the patient.

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EPIDEMIC PUERPERAL SEPSIS*

BY JOHN T. WILLIAMS, M.D.†

OUTBREAKS of puerperal infections occur from time to time in all large clinics, varying greatly in severity and in type in different years. The records of the Boston Board of Health show a regular curve in the incidence of respiratory infections from year to year, the peak months usually being March or April. Puerperal infections in the main follow a similar curve, as previously shown by the writer.[1] An outbreak of respiratory infection in the community is invariably followed by an epidemic of puerperal infection. Walker[2] of the Boston City Hospital and Meleney[3] of the Presbyterian Hospital in New York have shown essentially the same coincidence in surgical wound infections.

After many years of careful observation the writer has come to the conclusion that puerperal infections may be quite sharply divided into two groups: The first are mild infections, in which the temperature rises to 101° or 102° F. for from 1 day to several weeks and gradually returns to normal, and in which the patient is at no time seriously ill. These are simply wound infections analogous to mild surgical sepsis, due to avoidable or unavoidable breaks in technic, contamination from excreta or absorption from blood clots and serum. These infections are predisposed to by operative delivery, especially cesarean section, hemorrhage, toxemia and poor general condition of patient from any cause. Like the severe type of infection to be described, they are more common during outbreaks of respiratory infection. They are not serious, do not spread from patient to patient if ordinary care is used and do not necessarily require isolation.

The second group includes severe puerperal infections of the epidemic type. Here we are dealing with a disease as malignant as smallpox. This type is almost universally due to infection with *Streptococcus hemolyticus*. Although occasionally sepsis following full-term labor may be due to pneumococcus, staphylococcus or other organisms, the predominance of streptococcic infections is so great that for practical purposes other organisms may be excluded. In cases of septic abortion, which are not considered in this paper, the infecting organism may be almost any pathogenic bacterium.

The severe type of puerperal infection is characterized by a sudden rise in temperature to 103° or 104° F., 36 hours to 5 or even 7 days postpartum. The temperature remains elevated and the patient's condition usually grows rapidly worse. These infections usually occur only when there is an epidemic of respiratory infection present in the community. They are extremely contagious, being carried from patient to patient by hands, bedpans, perhaps dishes, in short, by anything handled or used by the septic patient and the nurse who cares for her. All infections occur more frequently after operative delivery than after normal delivery and most frequently after cesarean section.

Infection may enter the body through the placental site, cervical lacerations, perineal lacerations or the decidual surface of the uterus. The course of the infection from each of these four portals is as follows:

1. The placental site. When infection enters here direct transmission into the blood stream is common because of the large blood sinuses in this location and results in a true septicemia with positive blood culture.

2. Cervical lacerations. Infection here enters the cellular tissue of the broad ligament, often by way of the lymphatics, resulting in cellulitis. The blood culture is usually negative.

3. Perineal lacerations. These may occasionally give rise to severe infections, but as a rule the many layers of fascia tend to localize the process.

4. The decidual surface of the uterus. Here infection may be localized by a leucocytic zone with speedy recovery. Extension, however, may be by one of four paths: first, the blood, as in invasions of the placental site, resulting in septicemia; secondly, the lymphatics, resulting in cellulitis; thirdly, the fallopian tubes by direct continuity, resulting in salpingitis or occasionally local or general peritonitis; and, fourthly, the pelvic venous system, resulting in thrombophlebitis with or without secondary pyemia.

Where there is a localized pelvic process with a negative blood culture, the outlook for recovery is good. When the blood stream is invaded by the more virulent types of streptococci, the mortality is extremely high. No treatment seems to have the slightest effect. In our experience transfusions, single or repeated, do no good. Antistreptococcic sera, nonspecific protein therapy and outdoor treatment have made no appreciable difference in the mortality.

Dr. Champ Lyons[4] has recently published a study on transfusions with immune blood in hemolytic streptococcus infection at the Massachusetts General Hospital. He has found that

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certain individuals possess a relative immunity to such infections. If the blood from such an immune donor is used recovery may occur, while transfusion of blood from nonimmune donors has proved valueless. Such transfusions may prove to be of value in the treatment of puerperal sepsis.

Briefly described, his technic is as follows: The blood serum from several prospective donors is freed from cells by centrifuging. To 0.25 cc. samples of the patient's defibrinated blood are added 1 drop of each donor's serum and 1 drop of a broth culture of the streptococcus isolated from the patient. These are mixed in a sealed glass tube and slowly rotated for 30 minutes. A smear is made from the mixture, stained with Wright's stain and the number of intracellular streptococci in twenty-five polymorphonuclear leucocytes and the percentage of cells taking part in the phagocytosis is noted. The donor whose serum shows the greatest phagocytic power is selected for the transfusion.

Much progress has been made in typing streptococci by Lancefield[5] of the Rockefeller Institute, independently and in collaboration with Hare[6] and with Swift and Goodner.[7] Lancefield and her collaborators have worked out a classification of streptococci that divides them into seven groups which she designates by the letters A, B, C, D, E, F and G. Her classification is based on the fact that each of the different groups of streptococci contains a specific carbohydrate called the C substance.

Practically all streptococci pathogenic for human beings, such as those of scarlet fever, erysipelas, septic throat and pneumonia, belong in group A. They are also found in the throats of immune carriers. Groups B and C include the streptococci often found in human throats and vaginas that are pathogenic for animals but not for human beings. Group D is normally found in cheese and occasionally in the human throat and vagina. Group E, found in milk, and group F, found in the human throat, are probably not pathogenic for either animals or human beings. Group G, found in the normal respiratory tract in man, in the monkey in pneumonia and in the dog in otitis, is probably nonpathogenic to human beings.

It is not practicable as yet for the hospital with ordinary bacteriologic facilities to work out the pathogenicity of streptococci in the throats of its entire personnel or even in every case of infection. Lancefield has shown that the ordinary vaginal streptococci are nonpathogenic, so that the routine vaginal or cervical cultures offer little, if any, information of value.

While there is still some doubt as to the specificity of different strains of streptococci, the weight of evidence seems to favor the fact that at least the streptococci of erysipelas and of scarlet fever are specific. These are the erythrogenic streptococci. It would lead us too far afield to discuss this question further, but, as I shall show, what appears to be bacteriologically the same streptococcus produced in our clinic different manifestations in different years. For example, in 1933 our patients infected in the blood stream with *Streptococcus hemolyticus* lived from 15 to 95 days, while patients infected with what was apparently the same organism in 1934 died within 7 to 9 days.

Whether one makes the daily obstetric visit year after year or goes over the temperature charts of obstetric patients in retrospect, it is glaringly apparent that in the winter or spring of every year there is an epidemic of puerperal infection. At the Boston City Hospital we are on watch for it and try to isolate every case of infection as soon as it develops.

As I have previously stated, it has been definitely shown that the annual rise in respiratory infections is always followed by an increase in puerperal sepsis. The number of the cases and the severity do not necessarily correspond.

The material used in this study consists in the records of the deliveries and puerperia on the Obstetrical Service of the Boston City Hospital over a period of 8 years, 1926 to 1933 inclusive, a total of 19,315 patients. I have examined personally the records of every one of these patients and a large percentage were directly under my care. From the accompanying chart the incidence of mild and severe puerperal infections for the years 1926 to 1934 may be obtained. Of these years only one, 1928, was free from deaths from infection.

Starting in 1926, which was a mild year, there were few infections and only one death, although June and November showed an unusual number of severe cases. In 1927 there were a large number of mild infections amounting to 12 per cent of all deliveries, but fortunately with only 2 fatal cases. In 1928, as stated, there were relatively few infections and no deaths. In 1929, following a severe epidemic of respiratory diseases in Boston (see chart 1), there was a large incidence of mild infections, but severe cases were not relatively increased; there were three deaths. The year, 1930, was mild with few severe cases and only two deaths.

In 1931 there was one septic death in January. A severe epidemic began in April with 1 case following a normal delivery dying in 5 days from a *Streptococcus hemolyticus* septicemia. In May 5 cases of severe infection developed, among whom three deaths occurred, all after normal delivery. This epidemic continued into June with two severe infections, one following a normal and the other a low forceps delivery, both dying. After June severe infections dropped to an incidence of less than 1

per cent and no more deaths occurred until November.

In January, 1932, another severe epidemic started. It continued through February, but fortunately there were only two deaths. This epidemic was of especial interest because Kellogg and Hertig[8] have reported a similar epidemic, obviously throat born, occurring at the Florence Crittenton Home and starting the previous November, at which time, as above stated, we had a single death from infection. From March to December there were few severe cases; but, in December an epidemic began which continued through January and February of the following year.

The 1933 epidemic began in the preceding December. One death occurred in that month. In January and February there were 22 cases of severe puerperal infection with five deaths. In March there were 4 cases of severe infection and in April 5, but no deaths. In May, however, there were 6 cases of severe infection with one death.

An extensive bacteriologic survey was made by the Pathological Department. The only likely break in the aseptic chain was found to be a dressing table which, when not in use, was left near the entrance of the ward where it could have been contaminated by passing visitors. It is perhaps more important to note that this epidemic occurred in a period of extreme overcrowding due to the depression. January was the peak month for deliveries in the history of the hospital, a total of 369 women being delivered. The normal capacity should be about 150 patients per month.

All the fatal cases had positive blood cultures of *Streptococcus hemolyticus*. One patient died of streptococcus peritonitis 6 days after a normal delivery. The others lived, respectively, 15, 22, 79 and 95 days after delivery. In other words, this infection was almost what might be called chronic in type. This is in sharp contrast with the 1934 epidemic, in which none of the fatal cases lived over 18 days postpartum.

The 1934 epidemic was not included in the original study and is not charted, but, as it occurred shortly after this was completed and during my term of service in the hospital, I was able to observe the course of the entire epidemic. It differed from that of the preceding year in that the duration of the illness to a

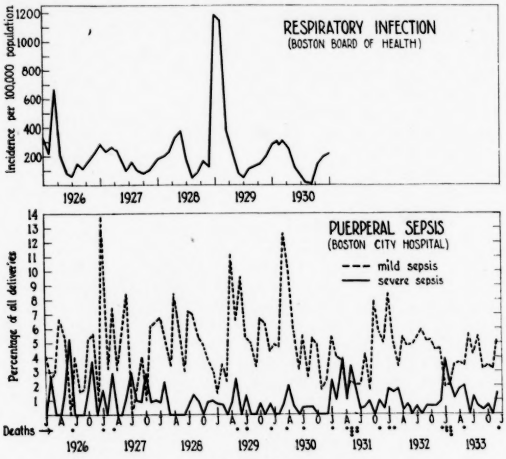

CHART 1.

This chart shows the incidence of mild (in broken line) and severe (in continuous line) puerperal infections on the obstetrical wards of the Boston City Hospital during the eight years, 1926 to 1933, inclusive, and the incidence of respiratory infections in Boston for the years, 1926 to 1930, inclusive. Note that the peaks of both mild and severe infections usually occur in the late winter and early spring and usually follow quite closely the curve of respiratory infections. The curve of the severe infections either coincides with or follows closely that of the mild infections. Note also the variation in severity of the puerperal epidemics from year to year; 1931 and 1933 were the worst years and 1926 and 1928 the mildest. Deaths from puerperal infection are indicated by the dots below the base line.

fatal termination was much shorter, although bacteriologically the same organism appeared to be responsible.

This epidemic started in January with a patient delivered by cesarean section for placenta praevia who died on the eighth day of a *Streptococcus hemolyticus* peritonitis. This patient was delivered on January 7. Two patients delivered on January 9 developed severe infections, but both had negative blood cultures and recovered.

There were no further severe infections until February, the last two mentioned patients having been discharged in the meantime. On February 8 a multipara was delivered normally, but some difficulty was experienced with the placenta. There was a rise in temperature on the fourth day to 104° F. Exploration showed a piece of retained placenta in the uterus, which was removed with placental forceps. The patient, however, developed a *Streptococcus hemolyticus* blood stream infection and died 18 days postpartum.

Three other patients, one delivered February 13, two on February 14 and all sent to the same ward, developed severe infections. High temperatures developed on the sixth, third and fourth days, respectively. Two had positive blood cultures and died. The third, although very ill, had three negative blood cultures and recovered. There were no further infections, due, I believe, to the fact that after the first case developed all other patients were promptly isolated. There can be little doubt that the second, third and fourth patients received their infections from the first, and as soon as a policy of prompt isolation of all cases of suspected infection was adopted the epidemic was brought to a close.

The Pathological Department was again called in consultation and, after an extensive bacteriologic study of catgut, dressings, instruments, sterilizing solution and throats of nurses and interns, found spore-bearing bacilli in the sterilizing solution and hemolytic streptococci in the throats of two nurses. The nurses were relieved from duty at once and a check-up on the sterilizing solutions at frequent intervals was instituted.

CONCLUSIONS

1. Puerperal infections may be divided into simple wound infections and epidemic puerperal infections usually due to *Streptococcus hemolyticus*.

2. Operative vaginal delivery and more especially cesarean section greatly increase the incidence of all types of infection. The mortality is practically the same for operative and non-operative deliveries, but much higher after cesarean section (table 1).

Type of Delivery	Total	Mild Sepsis	Severe Sepsis	Deaths
Normal	14,830 (76.8%)*	462 (3.1%)†	72 (0.5%)†	16 (0.1%)†
Operative vaginal	3,944 (20.4%)	173 (4.4%)	56 (1.4%)	4 (0.1%)
Cesarean section	541 (2.8%)	125 (23.1%)	25 (4.6%)	6 (1.1%)

*Percentages of total deliveries.
†Percentages of deliveries of that type.

TABLE 1. This tabulation shows the numbers and percentages of normal and operative vaginal deliveries and cesarean sections and of mild and severe sepsis and deaths following each type of delivery at the Boston City Hospital for the years 1926 to 1933, inclusive. Note that the percentages of septic deaths are the same for both normal and operative vaginal deliveries, but that the percentage of death is much greater following cesarean section.

3. Ordinary, or even extraordinary, aseptic precautions are not sufficient to guard against the epidemic type of infection.

4. No treatment, except possibly transfusion of immune blood, is of any value in epidemic *Streptococcus hemolyticus* infections.

5. Immediate isolation of all patients showing symptoms suggestive of severe puerperal infection is the chief means at present of preventing the development of epidemics.

6. Culturing the throats of the staff, interns and nurses is not of much value unless the hospital has bacteriologic facilities for typing the streptococci that are found.

7. Careful masking of all delivery room attendants is essential, although it is admitted that most of the present-day masks are inefficient.

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DISCUSSION

DR. ROBERT NYE: There is little I can say from a clinical point of view in a discussion of Dr. Williams' excellent paper. He has made reference to certain facts which are most interesting to the

bacteriologist and I thought that it might be worth while to consider these in more detail.

During the past two years Hare of England has published numerous papers that have contributed a great deal to our knowledge of the bacteriology and epidemiology of puerperal sepsis. Hare, a trained bacteriologist, became interested in the fact that, although hemolytic streptococci could be isolated from the vaginas of from 5 to 6 per cent of all cases before delivery, many, in fact most, of the cases failed to develop sepsis. He showed that the strains isolated from cases that remained afebrile were readily killed by freshly drawn normal human blood, whereas the strains from cases of puerperal sepsis remained alive and multiplied in such a medium. In a second paper, he reported that strains isolated from acute infections of the upper respiratory tract were identical in their behavior in human blood with the virulent vaginal strains; and, in a third paper, he proved that the virulent and avirulent strains could be further differentiated on the basis of certain biochemical reactions.

About this time, Lancefield's work appeared; and, as Dr. Williams has told you, she was able to divide all streptococci into seven serologically distinct groups of which the first, group A, contained the majority of strains that were pathogenic for human beings. Employing Lancefield's technic, Hare reclassified his strains with the following results. Of 45 strains from cases of severe sepsis, 44 were group A. From 837 normal hospital deliveries, hemolytic streptococci were obtained from 85 cases postpartum, but only 2 were group A. One of these cases developed a fatal sepsis, while the other remained afebrile. From 855 normal "district" deliveries, hemolytic streptococci were obtained from 13 cases antepartum. None fell into group A and no case developed severe infection.

In his two most recent papers he has shown that group A streptococci can be obtained from the noses and throats of about 7 per cent of all normal individuals and that such strains can be recovered from the stools of about 20 per cent of all nonobstetrical cases with severe streptococcic upper respiratory infection (scarlet fever), although they are never found in stools from cases with normal deliveries.

From these facts it seems reasonable to conclude: 1. Infective (group A) strains are only rarely encountered in antepartum vaginal cultures or in postpartum cultures from afebrile cases. 2. Infective strains are identical with those causing upper respiratory infection. 3. Infective strains are implanted in the vagina at or after delivery. 4. The sources of such infective strains, in the order of their frequency, are—(a) contact infection from the nasal or oral discharges of attendants, (b) cross infection from the nasal, oral or vaginal discharges of other patients and (c) auto-infection from the nasal or oral discharges or feces of the patient.

These clear-cut findings are, in my mind, somewhat complicated by our belief at the bacteriologic laboratory of the Boston City Hospital that we can differentiate two types of hemolytic streptococci. The first of these is the true, beta hemolytic type which produces a definite, "punched-out" zone of hemolysis when cultured on the surface of sheep's blood agar. As you all know, a colony of the alpha type streptococcus, *Streptococcus viridans*, is surrounded by a zone of greenish discoloration, presumably due to the formation of methemoglobin. The second type of hemolytic streptococcus, on primary isolation, has a colony which is intermediate between the true beta and the true alpha, and for this reason we have called it atypical alpha or alpha prime—the latter name should not be confused with that proposed by Brown of Baltimore. Surrounding the colony is a zone of true beta hemolysis, but this is bounded peripherally by a narrow zone of alpha hemolysis. After two to five subcultures on blood agar the latter characteristic is lost and the colony becomes typical of a true beta hemolytic streptococcus.

In 59 cases with positive blood cultures for streptococci received, during 1935, from all services of the hospital, 34 strains were classified in the laboratory as true beta and 25 as atypical alpha or alpha prime. Examination of the clinical records has yielded the following facts. Of the 34 cases with true beta infection, 26 died—a mortality of 77 per cent. The fatal cases had an average age of 49 years and an average duration from the first positive blood culture to death of 3.8 days. The recovered cases had an average age of 14 years with a maximum of 28 years. Furthermore, 24 cases were admitted during the first four months of the year, 2 during the middle four and 8 during the last four. Of the 25 cases with atypical alpha or alpha prime infection, 11 died—a mortality of only 44 per cent. The fatal cases had an average age of 41 years and an average duration of 11.7 days. The recovered cases had an average age of 29 years with a maximum of 50 years, and the incidence of all cases during the three four-month periods of the year were 10, 7 and 8, respectively.

These clinical facts bear out our assertion that there are two different types of hemolytic streptococci. It would seem to appear true that the atypical alpha or alpha prime streptococci are less virulent, as measured by mortality and by duration of illness, and do not have a seasonal incidence, like the true beta type, which parallels that of acute upper respiratory infections. At present we are saving strains of this type with the idea of determining whether they belong in Lancefield's group A.

I have mentioned this apparently less virulent type of streptococcus because a relatively large percentage of infections on the obstetric and gynecologic services are due to this organism. Of the 34 cases last year with bacteriemias due to beta hemolytic streptococci there were only 3 (less than 10 per cent) from these services, whereas of the 25 cases with bacteriemias due to the less virulent type 7 (over 25 per cent) were from the obstetric and gynecologic services. Furthermore, two of the epidemics of which Dr. Williams has spoken were due to streptococci of the less virulent type. If I remember correctly there were, in one of these (1932-33), 8 cases with positive blood cultures. One case died fairly acutely, 4 died after an illness of from 2 to 13 weeks and the other 3 recovered. The eventual mortality rate in this epidemic was exceptionally high.

From the bacteriologic point of view, the following precautionary measures against puerperal sepsis seem indicated: 1. Weekly nose and throat cultures of all attendants with exclusion from attendance of all with positive cultures, unless the strain is proved not to be a group A streptococcus. 2. Exclusion from attendance of all with acute upper respiratory infections until nose and throat cultures are negative. 3. Nose and throat cultures of all patients on admission and subsequent isolation of those with positive cultures. 4. Proper sterilization of bedpans, hot water bags, packs, instruments and so forth.

In conclusion, I will outline the procedures that are to be recommended in the event that a case of puerperal sepsis develops on a ward. 1. Immediate isolation of the case. 2. Nose and throat cultures of all attendants, with exclusion from attendance of all showing positive cultures. 3. Nose, throat and lochia cultures of all patients, with isolation of those with positive cultures. Do not blame the catgut or dressings! It is much more apt to be a

streptococcus from the nose or throat of the Obstetrician-in-Chief.

DR. CHAMP LYONS: The remarks of Dr. Williams and Dr. Nye must make it evident that the intelligent management of hemolytic streptococcus infections demands the special attention of a bacteriologically competent clinician. For the past three years I have been attempting to establish such a department for the study of surgical infections at the Massachusetts General Hospital.

We have had the opportunity of examining positive hemolytic streptococcus blood cultures from some 150 patients. The cultures have been made on horse blood agar, and we have not recognized the alpha prime type described by Dr. Nye and recognized by using sheep's blood agar plates. However, all the types which have shown true beta hemolysis on horse blood have been found to resist phagocytosis in nonimmune blood and to develop capsules. I am hopeful that we have worked with some of the alpha prime type in this group of positive cultures and that they will fall into the same general group for purposes of working with them in the phagocytic test.

Dr. Williams has indicated a seasonal prevalence with annual variation in the incidence of streptococcus infections. During the summer months we are accustomed to finding the "F" variant of streptococcus which is virulent for man and not for mice. As winter comes on there appears the "M" variant which is primarily virulent for both man and mice. In 1934, the "M" variant first appeared in January; in 1935, February; but this year we saw it in September. I have a feeling that the early appearance of the "M" variant in throat cultures, abscesses and blood cultures presages a dangerous streptococcal season.

Dr. Nye has indicated a difference in mortality between the earlier and later decades of life. In studying the natural development of phagocytic antibodies in patients with lympangitis and cellulitis, it would appear that young individuals develop demonstrable antibodies in the blood about the fifth day. In older individuals these are not demonstrable until the eighth or ninth day.

I have attempted to treat patients with positive beta hemolytic streptococcus blood cultures by transfusing them with blood from donors that shows a high titre of phagocytic antibodies for the specific type of streptococcus which is causing the infection. In the small series of patients that we have treated we have been able to clear the blood stream of the organisms unless there was an underlying endocarditis or septic phlebitis. Within 36 hours after such a transfusion there has been definite clinical improvement with evidence of localization of the infection. This is consistent with what has been obtained by passive immunization with antibacterial antibodies in other infections. It should be borne in mind that sterilization of the blood stream may be followed by the development of a local lesion in some vital center which may of itself be fatal. The series of cases treated is as yet too small to permit any critical evaluation of this method of treatment, but the underlying immunologic considerations are so well established that it is an entirely sound procedure.

The surgeon must accept the responsibility for the prevention of streptococcus infection. Virulent streptococci are harbored chiefly in the nasopharynx. Patients are infected by droplets through the air or indirectly by hands that have been contaminated by such droplets. It is possible that we have not given enough thought to the patient's throat and hands as a source of infection. Our treatment of acute burns has lately been modified to include face masks for the patient, as well as for the attendants, until such time as throat cultures have been obtained on the patient. We also make the effort to keep the patient's hands away from the nose and throat and to keep their hands washed after every such contact. It should be emphasized to nurses and attendants that their hands should be washed after every contact with their nose or throat. They do not realize that they may contaminate their hands by simply putting their hands to their mouths when they cough or blow their noses.

At the present time, prevention of infection is solely a matter of exclusion. To exclude all who have hemolytic streptococci in their throats would mean a reduction of about one-third of any given hospital staff during the winter months. It would appear from certain studies recently reported that it may be possible to isolate from such a group of positive throats a much smaller group by establishing the virulence or avirulence of the streptococci present in their throats.

HOME NURSING

BY ALFRED WORCESTER, M.D.†

THE training of nurses began in this country in 1872.* During the next few years nursing schools multiplied amazingly. Their graduates were largely employed in organizing new schools. The hospitals were thus soon supplied with pupil nurses and the economic advantage of such service, as well as its excellence, was a large factor in the rapid increase of American hospitals.

The redemption of hospital nursing from such incompetence and degradation, as now is hardly conceivable ranks only second to the introduction of asepsis in the marvelous advance of hospital service in the last half-century. What more then could be asked of the nursing schools? Well, let us consider this question.

Dazzled by the brilliant achievements in hospital nursing, it was not until recent years that the graduates of these schools realized the insufficiency of such training for nursing service in other fields. Then this recognition of what earlier had been clearly seen in England led to the establishment of American training courses for district visiting and public-health nursing. In these specialties the nursing profession has since then made notable progress. But in home nursing there has been no corresponding advance. This sidetracking of an equally important specialty has been due primarily to the neglect of training for such serv-

*At the New England Hospital for Women and Children, where Linda Richards in 1873 received the first American Nursing School diploma.

†Worcester, Alfred—Henry K. Oliver Professor of Hygiene Emeritus, Harvard University. For record and address of author see "This Week's Issue," page 1043.

ice. Just as hospital training was formerly considered sufficient for visiting nursing, for which special training is now considered indispensable, so the same mistaken idea persists that hospital training suffices for home nursing.

As a result of this misconception there are now thousands of graduate nurses without full employment while countless more thousands of home patients are without the needed nursing care. Nor is this true only of the poor and of families having slender means. To no small extent it is also true of those who could well afford to pay the prevailing wages of graduate nurses and would willingly do so could they find nurses who are expert in home nursing and really like it. Such nurses are scarce.

The young graduates of the hospital schools are fully able to transform homes into temporary hospitals. Sometimes this is what is needed for the patient. But such exigencies rarely occur. In the great majority of cases such impromptu hospitalizing is what is neither wanted nor needed. In this usual run of cases the nurse who has had only hospital training is from the start at a great disadvantage. She does not even know what is wanted. For example, in the hospital wards the visits of her patient's family, though limited to certain hours of certain days, were tolerated as unavoidable interruptions. How can a nurse with only such training realize that in the home her duty is to share with the family the care of their patient? In the hospital she has learned how to take care of isolated individuals. But in the home where the patient is one of the family a very different kind of nursing is needed. And, for this service, special training even if not absolutely essential is at least of enormous advantage both to the nurse and to the family she serves. This fact is not so generally recognized as it should be even by the medical profession. This is strange, for physicians well know the disadvantages of inefficient nursing in their patients' homes.

Unless the doctor can be sure of having his orders intelligently and faithfully executed, what chance is there for the success of his treatment of the case? And without an adequate supply of nursing service upon which they can rely is it any wonder that doctors nowadays often favor the removal to the private hospitals of patients who, with proper nursing, would do just as well and certainly be more comfortable if left in their own homes?

Like every other art, the art of nursing patients in their homes can be learned in the hard school of experience. But it can be learned far more easily and perfectly by imitation, that is, by practice under teachers who are themselves masters of the art. If this fact be admitted, it necessarily follows that, before undertaking home nursing, nurses should have the opportunity for special training in this specialty. Upon this conviction the Waltham Training School for Nurses was founded, and for half a century it has struggled against the contention of the leaders and organizations of American nurses that hospital training is sufficient for the practice of all other kinds of nursing.

Some twenty or more years ago, when seeking for Waltham nurses the privilege of registration in New York, the School paid for a survey by one of the official examiners of training schools for the New York Regents. Her main criticism was that if Waltham patients were not so well supplied with nursing service in their own homes more of them would have to go to the hospital, thus affording larger opportunity for the hospital training of our pupil nurses. Such rejection of our ideals would have been still harder to bear had not the School been following Florence Nightingale's advice and receiving her approbation. And as our preliminary course for probationers and our training course in visiting nursing had finally been adopted by other schools, we continued hoping that our belief in the necessity of special training for home nursing might also be accepted by the leaders of American nursing education. But with the continued increase in the educational requirements for state registration, which already embarrasses the hospital schools, such hopes grow less likely of fulfillment. The more probable alternative seems now to be that such training will have to be given in postgraduate courses just as special training now has to be given for visiting and public health nursing.

The hindrances to such a development are obvious. For as yet there are no organizations to ensure regular wages and free hours for nurses who are engaged in home nursing. But the main hindrance to such postgraduate courses is the general distaste of graduate nurses for home nursing. This in large part is due to their not having been taught this specialty. Very naturally nurses like best the services for which they have been best trained. More than that, it is a fair question if hospital training does not actually tend to unfit the nurse for home nursing. Even if in her own home she has been taught the art of housekeeping, in the hospital such lessons often seem to be unlearned. In the home, for examples, a towel after once used for drying well-washed hands does not have to be relaundered; the lighting and heating of the house also have to be conserved; all food waste has to be avoided; and all sorts of makeshift material have to be used. But the inconvenience of such necessary household economies is as nothing compared with the necessity of working *with* the family in the case of *their* patient. Surprising as it may seem to the nurse, the rough

hands of the farmer may convey to his sick wife or child far dearer comfort than massage by strange hands however softer and more skilful.

Perhaps the most important thing for the hospital nurse to unlearn is the habit of commanding. Ward maids and orderlies can be ordered by the nurses to do this and not that. Not so can be the household servants. Nor can members of the family be reminded of their ignorance without chilling their cooperation. Indeed, in home nursing one of the requisite services of the nurse is that of teaching the family how to take care of their patient. Such possible extension of the art of nursing, by tactful instruction of the family, serves not only their present but also their future needs. And the adept in home nursing must therefore have learned a higher joy than that of personal prowess, namely, the joy of seeing her patient perfectly nursed, under her instruction, by those most dear to him. In fact this ability of the nurse to keep herself in the background, which never can be learned in the hospital wards, is, nevertheless, for home nursing an angelic accomplishment. The list of essential qualifications for excellence in home nursing might easily be extended beyond our present limits. But perhaps enough has been said about the necessity of special training *in* home nursing *for* such service. Two further questions await discussion. First: What need is there for more home nursing than is now available, and, secondly, how can this need be met?

Deplorable as it may seem to modern doctors and nurses, there are still many folks who would rather have their babies born in their own homes than in a hospital, and so too when they themselves or those dearest are sick, even if near to death, these unconverted people (who, by the way, constitute nine-tenths of our population) still prefer home care however inefficient it may be if compared with that given in the best of hospitals. This is more true of those who live in impoverished homes than it is of those who live in modern palaces. But only a small proportion of these home loving families can afford to pay the prevailing wages of trained nurses. In the cities and larger towns much help can be had from the short visits of the District Nurses; and in the sparsely settled country neighbor-nursing is still available. And yet only a casual survey is needed to convince anyone that in the homes of our people there is a tremendous need for more and better nursing for the helpless sick and suffering.

How can this need be met? The short answer to this important question is, "By organization of all existing and prospective nursing power." This of course needs amplification. In the first place it is to be noted that in only a fraction of the total needs of home nursing is there need of fully trained nursing service. Just as in the hospitals, where far the greater part of the nursing is given by pupil nurses, so, and with the same economic advantage, a large part of the needed home nursing might be as perfectly supplied by nurses in process of training for the practice of this specialty. Such use of pupil nurses by many visiting nursing organizations affords a still more apposite example for similar organizations for home nursing which could easily be established even in our villages. Far easier it is to start such an organization, and less costly for any community will be its maintenance, than the starting and maintenance of a small hospital. Both are needed, but the organization for home nursing is needed first.

With a few well-trained nurses on salary, to serve where such service is essential, who will also superintend the service of pupil nurses and that of untrained neighbors wanting such employment, such an organization as we here have roughly outlined could begin tomorrow in any community. For all the service thus given the beneficiaries, if able to pay, must of course pay full charges; but less or nothing if such reductions are needed. The cost, above its earnings, of carrying on this work ought of course to be provided by family insurance against the possible need of medical and nursing service. But until the excellent Brattleboro plan is more generally adopted the cost of adequate home nursing must be provided by good neighborliness for those unable to pay for it.

In any community, or at any rate on any nursing registry, it will not be hard to find nurses who in one way or another have become proficient in the art of home nursing. Even if in demand by families they have previously served, such nurses may be very willing to accept permanent homes and regular salaries in a nursing service organization. But it may not be such an easy matter to find nurses who are also able and willing to teach their art to others. There is therefore need of schools for graduate nurses where both the art of home nursing and the art of teaching it shall be taught. Such a course the Waltham Training School for Nurses is now offering.

VERMONT STATE MEDICAL SOCIETY

HOUSE OF DELEGATES

THE Preliminary Meeting of the House of Delegates of the Vermont State Medical Society was called to order by the President, Dr. F. J. Lawliss, at eight o'clock, Wednesday evening, October 14, 1936, on the roof garden of the Hotel Vermont, Burlington, Vt.

President Lawliss appointed as the Credentials Committee: Dr. E. A. Hyatt, Chairman; Dr. R. E. Avery and Dr. C. G. Schurman, and requested members to present their credentials to this committee.

The credentials committee reported 31 delegates present, out of an authorized 38 delegates.

The Secretary, E. J. Rogers, called the roll. Those present were as follows:

ADDISON COUNTY MEDICAL SOCIETY:	
F. C. Phelps, Vergennes	
C. S. Paine, Bristol	2
BENNINGTON COUNTY MEDICAL SOCIETY:	
None present at this session.	
CHITTENDEN COUNTY MEDICAL SOCIETY:	
H. E. Upton, Burlington	
H. A. Durfee, Burlington	
J. L. Berry, Richmond	
W. F. Rogers, Underhill	
C. H. Beecher, Burlington	
E. D. McSweeney, Burlington	
G. I. Forbes, Burlington	
F. J. Arnold, Burlington	8
FRANKLIN COUNTY MEDICAL SOCIETY:	
L. E. Sample, St. Albans	
E. A. Hyatt, St. Albans	
F. J. Lawliss, Richford	3
LAMOILLE COUNTY MEDICAL SOCIETY:	
B. L. Emerson, Johnson	1
NORTHEASTERN COUNTY MEDICAL SOCIETY:	
M. J. Paulson, Danville	
P. C. Templeton, Irasburg	
C. G. Schurman, Newport	
W. A. Sargent, Newport	
E. T. Wilson, St. Johnsbury	5
RUTLAND COUNTY MEDICAL SOCIETY:	
Francis Quigley, Rutland	
E. J. Rogers, Pittsford	
B. F. Cook, Rutland	3
WASHINGTON COUNTY MEDICAL SOCIETY:	
C. E. Brady, Barre	
W. R. Harkness, Montpelier	
R. E. Avery, Barre	
L. B. Allen, Post Mills	4
WINDHAM COUNTY MEDICAL SOCIETY:	
R. E. McSweeney, Brattleboro	
O. A. Burton, Westminster	2
WINDSOR COUNTY MEDICAL SOCIETY:	
Paul C. T. Bacon, Springfield	1
	29

President Lawliss stated that, this being the first year that the House of Delegates has met prior to the opening of the scientific sessions of the Vermont State Medical Society, the meeting would be conducted in the same manner as the regular meetings of the House of Delegates had been in the past.

Article 3 of the Order of Business, the Secretary's report, Dr. E. J. Rogers stated would be presented at a later time, it being in the possession of the Secretary of the Vermont State Medical Society.

Article 4, Reports of Committees, were taken up, the first report called for, being that of the Publication Committee. Supplementing the printed report, Dr. W. G. Ricker, Chairman of this Committee, stated that the committee had continued, as in the past few years, the arrangement of the Society with *The New England Journal of Medicine*, and added that later in the evening more would be said about this.

The report of the Executive Committee was called for, and Dr. Ricker, Secretary of the Society, stated that the program for this year's Society meeting was the report of this committee.

President Lawliss called for the report of the Legislative Committee. Dr. C. H. Beecher, Chairman of this committee, stated that there was no printed report, and added: "The committee attended the session of the legislature in Montpelier on the day when there was a hearing on the bill before the Senate to license naturopaths; that the bill was eventually killed after having passed the Senate; that Doctors Tobin and Quinn, both members of the legislature, did very effective work in seeing that the bill did not pass. The argument against its passage, as presented at the hearing, was that the bill was unfair to the students who are studying medicine in that the requirements for admission to the practice of naturopathy were less than for the study and practice of medicine."

The report of the Committee on Medical Education was called for, and it was stated that there was none further than what had already been printed.

The report of the Necrology Committee was read, following which the House stood in one minute's silence as a tribute to deceased members.

President Lawliss called for the report of the Medico-Legal Committee, and Dr. Ricker stated that it had been sent to him but for the time being it had been mislaid. Dr. Hyatt, Chair-

man of this Committee, stated there had been one lawsuit that went to trial, and resulted in a verdict against the defendant. Several other minor cases were pending.

President Lawliss called upon the Secretary of the House of Delegates, Dr. E. J. Rogers, to read a communication from Dr. J. N. Jenne.

Secretary Rogers read the following communication:

Burlington, Vt.,
October 2, 1936.

Honorable House of Delegates,
Vermont State Medical Society

Gentlemen:

I am hereby tendering my resignation as a member of the Medico-Legal Committee of this Society after a period of incumbency of about twenty-five years. I take this opportunity of expressing my deep appreciation of the confidence of the Society during this long period. I feel that I should now be excused from this service which has been performed willingly, happily, and for some reasons, I feel, successfully. I think, however, that the time has come when a little new blood should be infused into the Committee.

As I review the work of the Committee from its beginning through these long years, it does seem to me that we have made some very distinct advancement. We have had a very fortunate experience in the last few years. I cannot but feel that it is due, in a large measure, to the attitude that the Committee and the Society have taken in medico-legal cases—that being one of absolute fairness in all cases where there was a justifiable claim against the practitioner, and one of firmness against all claims that seemed unfair or unjust to the doctor. The reports of the Secretary of the Committee and the Treasurer of the Society will disclose the present status of the Committee with respect to pending cases and the state of our fund.

Very respectfully submitted,
J. N. JENNE, M.D.

President Lawliss stated that Dr. Jenne and Dr. Hyatt had both served on the Medico-Legal Committee ever since its inception, and that the committee was originally formed at the suggestion of Dr. Hyatt.

Dr. Hyatt moved that the House of Delegates, through its Secretary, express to Dr. Jenne the thanks and appreciation of the Society for the fine work he had done on this committee during the past twenty-five years. The motion was seconded, and so voted.

President Lawliss called for the report of the Committee on Health and Public Instruction. No report was presented.

President Lawliss asked if there were any other committees that wished to report at this time.

In response to this Dr. Beecher said: "There is one committee that was asked to find out about the cost of sending *The New England Journal of Medicine* to each member of the Society once a week instead of once a month." Secretary Rogers said that matter would be taken up in the report of the Secretary of the Vermont State Medical Society.

Under Article 5, Unfinished Business, none was presented.

Under Article 6, New Business, (a) Place of next Annual Meeting, it was stated that invitations had been received from Bennington and St. Johnsbury. There being no objection, the President referred this to the Executive Committee to decide. Under (b) of this Article, "Other Business", Dr. Ricker, Secretary of the Vermont State Medical Society, was given the privilege of the floor and read his report as printed, going into detail on some of the matters mentioned therein, and adding that the publishers of *The New England Journal of Medicine* had offered to send 52 issues of the *Journal* for three dollars additional.

Dr. Beecher moved that the President and Secretary of the Vermont State Medical Society, also the President-Elect of the American Medical Association, be allowed to sit in this meeting of the House of Delegates. The motion was seconded and so voted.

Dr. Beecher moved that the House of Delegates express to Secretary Ricker its appreciation of the untiring labor he has performed in behalf of the Vermont State Medical Society over a period of some twenty years, and in making the Society an efficient unit of the American Medical Association. The motion was seconded and so voted.

It was moved that the report of the Secretary of the Society be accepted. The motion was seconded and so voted.

Proposed amendments to the By-Laws and Regulations of the House of Delegates were discussed at length, and President-Elect Upham, of the American Medical Association, was asked to outline the practices of that organization.

Dr. Upham stated, in substance, that delegates and alternates to the American Medical Association are elected by state societies; that if a delegate cannot go he signs over his card to his specified elected alternate; if neither one can go then the matter is presented to the Secretary and President of the state organization, who recommend, or appoint someone to fill that place; that it is then taken up by the credentials committee and presented to the House of Delegates, which either authorizes, or does not authorize, the seating of that person, as the House sees fit, for legally only the elected delegate and specified alternate can represent their respective societies. It is always within the power of the House to seat anyone as an alternate.

In order to raise the salary of the Secretary of the Vermont State Medical Society, Dr. Hyatt moved to amend Section 3 of Article 1, in line 6, of the By-Laws of the Vermont State Medical Society by striking out the word "two" and

inserting in lieu thereof the word "four". (Page 29, printed "Constitution, By-Laws and Reports, Vermont State Medical Society, October, 1936.) This motion was seconded and so voted.

Dr. Hyatt moved that the amount allowed for the Legislative Committee be increased to a sum not to exceed $500, to cover the Committee's necessary expenses, the money to be paid out by the Treasurer on presentation of properly prepared vouchers. The motion was seconded and so voted.

On behalf of the Chittenden County Medical Society, Dr. H. A. Durfee offered the following resolution:

> **RESOLVED: That a committee of three be appointed by the Chair to confer with the Commissioner of Industries and representatives of the Vermont Manufacturers Association with a view to correcting the defects of the existing "Workmen's Compensation Act"—**
>
> **RESOLVED: That this committee be chosen as follows: one member from the roster of the House of Delegates, one member from the Vermont State Medical Society at large, and one member of the State Society who shall represent the interests of the Hospitals of the State.**

In connection with this resolution it was suggested that the Committee confer with the representatives of the Insurance Carriers and from them obtain all possible statistical information prior to the proposed conference with the Commissioner and the Manufacturers' representatives.

Dr. Beecher moved the adoption of this resolution as presented. The motion was seconded, and the resolution was adopted.

President Lawliss appointed as the committee of three, called for in this resolution, to confer with the Commissioner of Industries: Dr. E. A. Tobin, Chairman; Dr. L. E. Sample and Dr. T. S. Brown.

As a committee to put into proper form the various proposed amendments, and present the same at the regular meeting of the House of Delegates on Thursday, October 15, 1936, President Lawliss appointed Dr. C. H. Beecher, Chairman; Dr. E. A. Hyatt, and Dr. E. J. Rogers, stating that Dr. Ricker, the retiring Secretary of the Society, will sit with this committee. Dr. Rogers requested that the President of the House of Delegates also sit with this committee.

Under Article 7 of the Order of Business, the "Election of Officers for the Society", for the ensuing year, the Chair asked if the House of Delegates wished to make nominations from the floor or to refer this matter to a nominating committee.

Dr. Beecher moved that nominations for President and Secretary be made from the floor. The motion was seconded and so voted.

For President of the Society for the ensuing year, Dr. Hyatt nominated Dr. W. G. Ricker. The nomination was seconded and so voted.

For Secretary of the Society for the ensuing year, Dr. Beecher nominated Dr. A. Bradley Soule, Jr., of Burlington. The nomination was seconded and so voted.

It was then moved that the chair appoint a nominating committee to bring in nominations for the remaining officers and committees for the Society, and officers for the House of Delegates, at the regular meeting of the House on Thursday, October 15, 1936. The motion was seconded and so voted. The Chair appointed as such nominating committee Dr. E. A. Hyatt, Chairman; Dr. Hiram Upton and Dr. B. F. Cook.

There being no further business it was moved that the meeting adjourn. The motion was seconded and so voted, and the Preliminary Meeting adjourned.

The Regular Meeting of the House of Delegates of the Vermont State Medical Society was called to order by President F. J. Lawliss at one o'clock, Thursday afternoon, October 15, 1936, at the Fleming Museum, Burlington, Vt.

Upon vote of the members present, the following alternates were seated in place of the regular delegates:

WASHINGTON COUNTY:
Dr. H. B. Whiting for Dr. W. W. Angell.

BENNINGTON COUNTY:
Dr. F. J. Hurley for Dr. H. S. Goodall.

RUTLAND COUNTY:
Dr. Stewart Ross for Dr. O. F. Clough.

President Lawliss called for the reading of the minutes of the Preliminary Meeting of the House of Delegates, held Wednesday evening, October 14, 1936 at the Hotel Vermont.

Secretary Rogers stated that unless there was objection, he would not read these minutes in full; that the proposed amendments to the By-Laws and Regulations of the House of Delegates, and one proposed amendment to the By-Laws of the Vermont State Medical Society, had been referred to a committee composed of Dr. C. H. Beecher, Chairman; Dr. E. A. Hyatt and Dr. E. J. Rogers, to put them into proper form for presentation at this meeting.

For this committee Secretary Rogers read the following report:

Your Committee, appointed to put into form the proposed amendments to the By-Laws and Regulations of the House of Delegates, and one proposed amendment to the By-Laws of the Vermont State Medical Society and to submit the same to the Regular Meeting of the House of Delegates on Thursday, October 15, 1936, reports as follows:

That paragraph 1, of the By-Laws and Regulations of the House of Delegates, be amended to read as follows:

> 1. The officers of this body shall consist of a President and two Vice-Presidents, all of whom shall be elected from members of the Vermont State Medical Society. The Secretary of the Vermont State Medical Society shall act as Secretary of the House of Delegates.

That paragraph 2 of the "By-Laws and Regulations of the House of Delegates", be amended to read as follows:

> 2. The duties of Officers shall be the same as those of similar officers in other like organizations.

That paragraph 3, of the "By-Laws and Regulations of the House of Delegates" be amended to read as follows:

> 3. The first session of the House of Delegates, at each annual meeting of the Society, shall be held at 8 p. m. of the evening of the day preceding the first scientific session. The second session shall be held at one p. m. of the first day of the scientific sessions.

That a new paragraph, to be number 4, be added to the "By-Laws and Regulations of the House of Delegates" to read as follows:

> 4. No new business shall be introduced at the second session except by unanimous consent.

That a new paragraph, to be number 5, be added to the "By-Laws and Regulations of the House of Delegates" to read as follows:

> 5. No member of the House of Delegates, or his alternate, shall be seated unless certified by the President and Secretary of his County Society to the Secretary of the Vermont State Medical Society on or before September twentieth.

That a new paragraph, to be number 6, be added to the "By-Laws and Regulations of the House of Delegates" to read as follows:

> 6. (a) The Secretary of the Vermont State Medical Society shall furnish to each delegate a colored credential card containing his name and the name of his alternate.
> (b) The Secretary of the Vermont State Medical Society shall furnish to each alternate a white credential card containing his name and the name of the delegate for whom he is an alternate.
> (c) If a delegate is unable to attend the sessions of the House of Delegates he should notify his alternate.

Dr. Cook moved that all of the proposed amendments be adopted as read. The motion was seconded and was so voted.

Secretary Rogers read the following proposed amendment to the "By-Laws of the Vermont State Medical Society":

"To amend Section 3, of Article I, in line 6. by striking out the word 'two' and inserting in lieu thereof the word 'four'. (Page 29, 'Constitution, By-Laws and Reports, Vermont State Medical Society, October, 1936.')"

Dr. Beecher moved the adoption of this proposed amendment as read. The motion was seconded and was so voted.

Dr. Beecher moved that the Secretary of the Society be instructed to send a copy of these proposed amendments to the Presidents and Secretaries of the County Societies. The motion was seconded and so voted.

Dr. Cook moved that the Society pay the expenses of its delegate to the American Medical Association for the year 1936-1937. The motion was seconded and so voted.

The nominating committee submitted the following report:

"In addition to W. G. Rieker being elected President, and A. Bradley Soule, Jr., Secretary of the Vermont State Medical Society, the nominating committee submits the following list of nominees for officers, committees and delegates for the ensuing year:

VICE-PRESIDENT:
W. J. Upton, Burlington

TREASURER:
David Marvin, Essex Junction

AUDITOR:
Peter P. Lawlor, Burlington

COUNCILORS:
2nd District—G. G. Marshall, Rutland
4th District—A. M. Cram, Bridgewater

EXECUTIVE COMMITTEE:
A. B. Soule, Jr., Burlington
W. B. Fitch, St. Johnsbury
E. M. Cleasby, Orleans

PUBLICATION COMMITTEE:
A. B. Soule, Jr., Burlington
P. A. Goddard, Morrisville
H. E. Upton, Burlington

LEGISLATIVE COMMITTEE:
C. H. Beecher, Burlington
E. A. Tobin, Brattleboro
E. J. Quinn, Castleton

MEDICAL EDUCATION:
C. G. Abell, Enosburg Falls (3 years)
N. R. Caldwell, Burlington (2 years)
S. S. Eddy, Middlebury (1 year)

NECROLOGY COMMITTEE:
B. D. Adams, Burlington
G. G. Marshall, Rutland
H. L. Pierce, Swanton

MEDICO-LEGAL COMMITTEE:
E. A. Hyatt, St. Albans (1 year)
Stewart Ross, Rutland (2 years)
F. E. Farmer, St. Johnsbury (3 years)

HEALTH AND PUBLIC INSTRUCTION COMMITTEE:

B. F. Cook, Rutland
F. J. Lawliss, Richford
A. C. Black, Brattleboro
A. L. Patch, Windsor
W. W. Angell, Randolph

NOMINATIONS TO STATE BOARD:

F. J. Lawliss, Richford
B. F. Cook, Rutland
S. S. Eddy, Middlebury
Roland McSweeney, Brattleboro

DELEGATES:

Maine—John Stewart, Bellows Falls
New Hampshire—M. J. Paulson, Danville
Massachusetts—A. A. Cheney, Lyndonville
Rhode Island—A. L. Fogg, Burlington
Connecticut—John Trotter, Bennington
New York—Leon Sample, St. Albans

American Medical Association:
A. B. Soule, Jr., Burlington
C. C. Abell, Enosburg Falls, Alternate

ANNIVERSARY CHAIRMAN:

Frank E. Farmer, St. Johnsbury

OFFICERS OF THE HOUSE OF DELEGATES:

President, F. J. Lawliss, Richford
First Vice-President, Ralph Seeley, Rutland
Second Vice-President, E. D. McSweeney, Burlington
Secretary, A. B. Soule, Jr., Burlington

Dr. Beecher moved that the report of the nominating committee be accepted and adopted as presented. The motion was seconded and so voted.

The President declared the nominees named therein elected to the respective offices and committees for which they were nominated.

Dr. Beecher moved that the House of Delegates approve and adopt all of the work that was done at the unofficial Preliminary Session of the House held on Wednesday evening, October 14, 1936 at the Hotel Vermont.

The motion was seconded and so voted.

President Lawliss announced that the Executive Committee had decided to hold the 1937 meeting of the Society at St. Johnsbury.

There being no further business, Dr. Beecher moved that the House of Delegates adjourn. The motion was seconded and so voted.

Adjournment.

MISCELLANY

VERMONT DEPARTMENT OF PUBLIC HEALTH

SEPTEMBER, 1936

The following communicable diseases were reported to the office of the Department of Public Health during the month of September: measles 9; German measles 1; scarlet fever 10; chickenpox 17; mumps 21; whooping cough 107; undulant fever 3 and typhoid fever 2.

The Laboratory of Hygiene made 2,055 examinations, the details of which are as follows:

Examinations for diphtheria bacilli	71
" " Widal reaction of typhoid fever	54
" " undulant fever	80
" " gonococci in pus	164
" " tubercle bacilli	184
" " syphilis	768
" of water, chemical and bacteriological	212
" " water, bacteriological	263
" " milk, market	152
" " milk, submitted for chemical only	0
" " milk, submitted for microscopical only	5
" " foods	1
" " drugs	3
" for courts, autopsies	3
" " courts, miscellaneous	38
Autopsies to complete death returns	2
Miscellaneous examinations	55

The Director of the Division of Venereal Diseases reports 51 cases of gonorrhea and 75 cases of syphilis. Six hundred and fifty-three Wassermann outfits and three hundred and eighty slides for gonorrhea were distributed from this Division.

The Crippled Children's Division reports a total of two hundred and six patients seen, two hundred and thirty-eight home visits made, six calls on doctors and one hundred and eighty-five Social Service calls made. Four patients were admitted to the Audubon Hospital and one discharged. Five patients were admitted to the Children's Hospital and one discharged, and one patient was admitted to and discharged from the Massachusetts General Hospital. Eighty-four pieces of apparatus were fitted, forty-two orthopedic corrections to shoes were made and seven pieces of apparatus were repaired. The Vocational Worker of this Division reports sales for the month amounting to $351.36.

The Director of the Division of Public Health Nursing reports that most of the month has been spent in organization work. Two new units have been established, one in Addison County and one in Windham County. The WPA nursing project is still continued with thirty names on the payroll. Two hundred and sixty-one baby booklets, 1,080 diphtheria consent cards and 641 birth notifications were mailed during the month.

The Director of Maternal and Child Health reports an active month with many conferences and medical meetings attended. Dr. DeLee's Normal Labor Films have been shown at several training schools for nurses and three lectures were given as a part of the Refresher Courses in Obstetrics and Pediatrics.

ANNOUNCEMENT

Clarence F. Ball, M.D., announces the removal of his offices to 23 Washington Street, Rutland, Vermont, with John D. Southworth, M.D., as office associate.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 22481

PRESENTATION OF CASE

A 55 year old Norwegian asbestos worker was admitted complaining of pain in the abdomen and back.

For about 5 years the patient had occasional backaches caused by standing at work throughout the day. These were relieved by a night's rest. Nine months ago, however, he began to experience pain in the region of the lumbar spine which radiated up and down the back. The pain was aching and constant, rarely sharp and stabbing in character, and was not relieved by bed rest or change in position. It occasionally radiated directly anteriorly without encircling the body and was more severe in the left upper quadrant. It was frequently aggravated 3 to 4 hours after meals. Occasionally there was a sensation of heaviness in the stomach shortly after the ingestion of food. This was relieved initially by baking soda but for 2 months such treatment was unavailing. Bowel movements, following the administration of Epsom salts, and gaseous eructation not infrequently afforded some relief. His appetite became poor and there was occasional morning nausea and infrequent emesis after breakfast. Constipation was progressive during his illness and there was diminution of weight from 125 to 105 pounds. Associated with the weight loss there were weakness and fatigability. There was no diarrhea or melena.

Physical examination showed a well-developed, thin and weak-looking man with evidence of considerable weight loss. The skin was loose and dry but the color was normal. The fundi exhibited increased vascular light reflexes and some venous nicking but were otherwise negative. The vertebral column showed generalized limitation of motion without localized tenderness. The heart was not enlarged and its rhythm was regular. A systolic murmur was audible at the apex and the first sound in this region was reduplicated. The blood pressure was 160/110. The lungs were resonant and an occasional moist râle and coarse wheeze were audible at either base. The abdomen was scaphoid and there was a firm, nodular, tender, immovable mass which extended from the left costal margin to the level of the umbilicus and from the anterior axillary line to the midline. This did not move with inspiration. The percussion note over this region was dull but the remainder of the abdomen was negative. Slight tenderness was elicited in both costovertebral angles. Rectal examination showed a hard, slightly movable mass obtruding upon the posterior rectal wall but the prostate was not remarkable.

The temperature was 99.5°, the pulse 94. The respirations were 24.

Examination of the urine was negative. The blood showed a red cell count of 3,800,000, with a hemoglobin of 70 per cent. The white cell count was 9,040, 73 per cent polymorphonuclears. Repeated stool examinations were essentially negative and gave no reaction to the guaiac test. The serum protein was 4.8 per cent and the nonprotein nitrogen of the blood was 36 milligrams. A van den Bergh and a Hinton test were negative. The serum chlorides were equivalent to 90 cubic centimeters N/10 chloride per 100 cubic centimeters. Daily gastric aspiration showed 8 to 40 ounces of clear yellowish fluid with a slightly fecal odor. Guaiac tests of this fluid were negative.

X-ray examination of the back showed evidence of very marked hypertrophic arthritis. An intravenous pyelogram showed normal kidney pelves and ureters. The kidneys were likewise normal in size and shape. A chest study exhibited a low diaphragm but normal respiratory motion. There was slight mottling within the midlung field and the hilar shadows appeared slightly enlarged. There were multiple areas of calcification in both hila and in the peritracheal region. The heart and aorta were normal. Plain films of the abdomen showed a large amount of gas in the stomach but no dilated loops of intestine. A barium enema passed the ileocecal valve without delay. The palpable mass was not connected in any manner with the colon. A gastrointestinal series showed a normal esophagus. The rugae of the stomach were moderately hypertrophied but there was no other evident abnormality. The duodenal cap was large and showed increase in size of its rugae.

During the succeeding week the patient became unable to ingest food, vomited occasionally, and gastric aspiration withdrew increasing amounts of fluid and some gas. On the eighth hospital day a proctoscopy up to 15 centimeters above the anus showed a peculiar tapioca-like, glistening and translucent infiltration of the rectosigmoid. A rectal examination elicited the presence of a large hard mass the size of a plum which lay anterior to the rectum and displaced it backward. Subsequently the patient was treated palliatively and received fluids paren-

terally. He became progressively weaker and died on the eighteenth hospital day.

X-ray Interpretation

Dr. Aubrey O. Hampton: Here is the stomach and I have to accept the report that all it showed was thickening of the mucosa. The mucosa is definitely thickened and the stomach seems small and short, and it empties rapidly. It changes in shape. I do not think it is rigid. Here is a film of the gas filled stomach. It is larger there. (Indicating.)

The kidney outlines in the intravenous pyelogram showed nothing. I do not see the mass in the pelvis or any mass in the upper abdomen. We did not have a film of the colon with complete filling. This is a post evacuation film only. The rectum is in normal position. I do not see anything there. In all these films the abdomen looks dense, rather homogeneous, large and distended. The spine shows very little. I should say there are only moderately advanced hypertrophic changes instead of marked ones. The film of the esophagus and the lateral view of the chest are normal. His chest shows fairly definite evidence of pulmonary fibrosis and enlargement of the glands. He was an asbestos worker so probably it is asbestosis. Some of these deposits seem to be in the costal cartilages instead of the glands. In the lateral view, however, there are several quite definitely calcified glands, and this does not occur with asbestosis generally. This is not an advanced case. I can only say that the findings are consistent with asbestosis.

Differential Diagnosis

Dr. Arthur W. Allen: This is the kind of patient we would like to examine rather than to have an abstract of the history from which to make a diagnosis. The symptoms of his backache for 5 years I suppose can be accounted for on the basis of arthritis. The fact, however, that he had constant backache and pain radiating from back to front over a period of 9 months would make me feel that this pain was in some way to be accounted for by the mass, the palpable tumor. I guess there can be no question but that this mass is a malignant one with metastases to the pelvis involving the rectum secondarily, as evidenced by the fact that these tapioca-like projections could be seen in the proctoscopy, and that it could be felt by digital examination and was not ulcerative. A primary lesion in that region should be ulcerated by the time it reaches this stage and give blood in the stools. This man had negative stools, so I think we can assume that the implants in the rectum were secondary and not primary. The stomach being negative in the x-ray but nevertheless giving on aspiration large amounts of cloudy fluid with a fecal odor makes one feel that possibly there might be a lesion in the stomach that the x-ray men had not seen. We always hesitate to question their opinion because they are very accurate about their diagnoses.

The story is consistent with a stomach lesion, however, and the fact that he was relieved for a while with baking soda and various things suggests the possibility of an ulcerative lesion of the stomach that might later have become malignant. The laboratory data do not state whether he had any acid in his stomach. That would be somewhat helpful because if he had acid I think we could feel a little more certain that this was not carcinoma of the stomach. The fecal odor suggests the possibility of a spontaneous communication between the stomach and the colon. Gastrocolic fistula does occur in carcinoma of the stomach and in carcinoma of the transverse colon. We have had four instances of that in the last 10 years. I am familiar with these figures because I have been interested in gastrojejuno-colic fistula following gastroenterostomy. This man does not have a spontaneous fistula because if he did he would have had diarrhea and not constipation, and it would be a very easy lesion to pick up by the barium enema if the barium meal failed to show the fistula. The colon is probably not the site of the primary disease, because nearly always you can be certain of a lesion of this size. One that represents a palpable mass can be demonstrated by barium enema under the fluoroscope. I have known one exception in the transverse colon where the barium enema was entirely negative and the entire transverse colon was involved with carcinoma.

The small bowel has not been mentioned in the x-ray and apparently it did not make much impression on the roentgenologist when he went over it in the first place. But this tumor could be in the small bowel, a lymphomatous condition with pelvic metastases forming a movable mass in this region. We recently had such a patient with lymphoma involving the jejunum primarily as nearly as we could make out and later involving the descending colon, a mass that was as large as this one that is described. It could be moved and that patient also had pelvic metastases that could be felt by rectum. After the jejunal lesion and the sigmoid lesion were resected he was treated by x-ray and all his lymphoma has disappeared for the time being.

Dr. Hampton: I meant to mention the overfilling of the first and second portions of the duodenum and here you can interpret this defect as due to pressure from the spine and from a mass in front. This reduction in size of the stomach is possibly due to spasm or due to an extrinsic process, since we do not see it inside. I cannot say it is tumor of the pancreas but I

can say that it is consistent with it. I cannot see the tumor. There is no mass in the loop.

DR. ALLEN: A tumor of the pancreas would explain very adequately his backache and the gastric symptoms.

The probabilities are that this was not the spleen because it did not go clear back into the flank. The mass is very accurately defined from the costal margin to the umbilicus and from the anterior axillary line to the midline and does not go to the flank. Also, we can rule out hypernephroma to explain the mass on the basis that there was absolutely no blood in the urine at any time and also because the mass did not extend into the flank.

This case might, I suppose, be accounted for on the basis of tuberculosis. We have had at least one patient in this hospital with tuberculous mesenteric glands that produced a large mass; in this instance it was a young woman and the mass was thought by some of the men to be an ovarian cyst as it could be pushed into the pelvis and moved out of the pelvis. It was a mass in the mesentery of the ileum that was sufficiently large to require resection of about three feet of the ileum in order to get rid of the tumor. This man is rather old to have symptoms from calcified mesenteric glands.

I would put down as my first choice for diagnosis carcinoma of the pancreas with metastases to the pelvis, and, secondly, I should put lymphoma with rather generalized involvement of the retroperitoneal area as well as the peritoneal cavity. It probably is not a carcinoma of the stomach. I believe that if it were carcinoma of the stomach, as extensive as this, the stomach would be in a different location and look differently in the x-ray; also, I doubt if the mass would be quite so easily palpable if it were carcinoma of the stomach.

CLINICAL DISCUSSION

DR. CHESTER M. JONES: This patient was on the ward when I came on service and one of the most striking things was that he presented the symptoms of gastrointestinal obstruction. All of us felt that he had malignancy. It was a question of whether we were justified in advising exploration. The rectal mass was definite. The proctoscopy was the most curious one I have ever seen. It looked as if the entire wall were studded with soft tapioca. It did not bleed at all. I have never seen anything like it before. I raised the question whether it might be lymphomatous rather than metastatic malignancy from the gastrointestinal tract or the pancreas, but I think as we watched the patient's progress on the ward we felt that carcinoma of the pancreas was the most likely probability with pressure on the small bowel and almost complete obstruction before he died. The picture was one of intestinal obstruction. We all felt that exploration would serve merely to satisfy our curiosity and nothing else, and it was not done.

DR. TALBOTT: I saw this man the last few days before I went off service and at that time he presented quite a different picture than when Dr. Jones saw him. The picture was that of something outside the stomach. He did not give a history that made us think he had a lesion in the stomach. The only argument on the service was whether it was carcinoma of the pancreas or lymphoma. The masses were all fixed except the few nodules in the subcutaneous wall. The mass in the pouch of Douglas was a hard, firm mass which gave the feeling of something from the outside pressing in, rather than something in the rectum. Dr. Eugene Sullivan thought it was carcinoma of the pancreas. I thought it was lymphoma.

CLINICAL DIAGNOSES

Carcinomatosis, ? source, probably the body or the tail of the pancreas.
? Lymphoblastoma, retroperitoneal.

DR. ARTHUR W. ALLEN'S DIAGNOSIS

Carcinoma of the pancreas with metastasis to the pelvis.

ANATOMIC DIAGNOSES

Adenocarcinoma of the pancreas with extension to the stomach and duodenum and with metastases to the liver, visceral and parietal peritoneum, intestine, left adrenal and pericardium.
Pulmonary tuberculosis, healed.

PATHOLOGIC DISCUSSION

DR. BENJAMIN CASTLEMAN: The autopsy on this man showed that his abdominal cavity contained about 400 cubic centimeters of clear straw-colored fluid. The omentum and both the visceral and parietal layers of peritoneum were studded with large numbers of metastatic tumor nodules measuring up to a centimeter in diameter. Several were present on the undersurface of the diaphragm and the pelvis was filled with loops of small intestine adherent to each other and to the sigmoid and rectum by tumor tissue. There was, however, no ulceration into the bowel. In the region of the stomach, transverse colon and third portion of the duodenum was this huge fixed mass, about 12 by 12 by 18 centimeters, that compressed the third portion of the duodenum and produced a dilatation of the first and second portions. It was pretty hard to separate the colon and duodenum from the body of the pancreas but it was finally done by blunt dissection. The tumor was primary in the body of the pancreas and involved all but the distal 3 centimeters of the tail. There were huge metastases

in the liver and left adrenal as well as a smaller one, 2 by 1 by 3 centimeters, on the visceral pericardium. Microscopically the tumor is a fairly well differentiated adenocarcinoma. His lungs showed no evidence of asbestosis, but they did contain patches of bronchopneumonia. The smaller pulmonary arteries contained a few small adherent thrombi. There were no metastases to the bones.

CASE 22482

Presentation of Case

A 53 year old Finnish engineer was admitted complaining of pain in the right upper quadrant.

About one year ago the patient began to have attacks of pain in the right upper abdomen. These attacks persisted for about five minutes and the pain frequently radiated around and through to the right subscapular region. There was no nausea, vomiting, fever, relation to meals or associated change in bowel habits. There was no known precipitating cause except that bending forward occasionally initiated the pain. Four months before entry some shortness of breath with exertion was noted and there was slight increase in perspiration although he had no night sweats. Three weeks before coming to the hospital he had an attack of pain similar in severity to the others but persisting up to entry. He continued to work for a few days but finally was compelled to go to bed. A physician was called and hospitalization was advised. At this time the patient was informed that his sclerae were icteric. He went to a hospital for several days but left against advice. He remained at home in bed and the pain persisted with varying severity. For 2 weeks there was pain in both shoulders.

Physical examination showed a well-developed and moderately obese man with shallow, irregular, rapid breathing accompanied by coarse grunts. The skin was dry and there was questionable jaundice of the sclerae. The chest was barrel shaped but the lungs were clear. The heart was not enlarged. The sounds were of fair quality. A systolic murmur was audible over the precordium. The blood pressure was 130/85. The abdomen was distended and tympanitic. There were spasm and deep seated tenderness in the right upper quadrant. Rectal examination was negative.

The temperature was 100.2°, the pulse 90. The respirations were 24.

Examination of the urine was negative. The blood showed a red cell count of 4,700,000, with a hemoglobin of 95 per cent. The white cell count was 11,200, 77 per cent polymorphonuclears. Stool examinations were negative. The diastase index was normal. The nonprotein nitrogen was 26 milligrams and an icterus index was 5. A Hinton test was negative and an electrocardiogram was negative.

X-ray examination of the chest showed the heart to be normal in size and shape and the aorta to be slightly tortuous. Both hilar shadows were unusually large, particularly on the right side. The lung fields were clear. A Graham test showed no definite shadow of the gallbladder although several very faint suggestive areas were noted. The dye was seen in the colon. A gastrointestinal series showed no evidence of organic disease of the esophagus, stomach, or duodenum.

The patient's temperature fluctuated between 100° and 102°, his pulse between 90 and 110, and the pain was unabated. On the eighth hospital day he complained of pain in the right lower chest upon deep inspiration. At this time he coughed up about an ounce of blood-streaked sputum. Examination of the chest showed no dulness but a few fine râles were audible at the left base.

Another chest x-ray showed normal position and movement of the diaphragm and clear lung fields. There was a large mass in the right hilus with hazy density extending up along the right superior mediastinum. The mass was also seen in the lateral view and the trachea appeared to be slightly displaced to the left.

A red blood cell count at this time was 3,050,000, with a hemoglobin of 55 per cent. The white cell count was 14,300 and the smear contained 21 per cent band neutrophils, 39 segmented forms, 14 lymphocytes, 3 monocytes and 23 myelocytes. An icterus index was 9 and the van den Bergh 1.5 to 2 milligrams. A gastric analysis showed a free acid of 6 after ergamin and a total acid of 12. A serum phosphatase was 35 Bodansky units.

Another examination on the twenty-fifth hospital day showed a slight labial cyanosis. The right chest was dull to flat from the third to ninth interspace posteriorly and dull anteriorly from the third interspace down. Many expiratory wheezes were audible generally and breath sounds were diminished on the right side although the expiratory phase appeared to be prolonged. Fine moist râles were heard at both bases. The liver extended down about a handbreadth beneath the costal margin. Its edge was rounded and said to be smooth and nontender, although other examiners stated that it was hard, nodular and somewhat tender. The spleen was not felt, but deep in the left upper quadrant there was a tender indefinite mass which appeared to descend with respiration. There was tenderness in the left costovertebral angle.

A bronchoscopic examination was entirely negative but another chest x-ray showed increase in the size of the mass in the right hilus. There was now hazy dulness extending through the midportion of the right lung field just above

the septum between the middle and upper lobes. There was also a sharply defined mass projecting from the right upper mediastinum. X-rays of the spine and femora showed no evidence of metastasis and an intravenous pyelogram was negative.

Another white cell count was 14,700, 54 per cent polymorphonuclears, 20 band forms, 17 myelocytes, 3 myeloblasts, 3 lymphocytes, 1 eosinophil and 2 normoblasts. The patient continued to run a febrile course and still complained of severe pain in the right upper abdomen and back. There was also exquisite tenderness of the spinous processes of the lower dorsal and lumbar vertebrae. There was no further hemoptysis, but the patient became progressively weaker and died on the thirty-eighth hospital day.

X-ray Interpretation

Dr. Aubrey O. Hampton: This is the first examination. The mass described at the right lung root fades off gradually and the trachea is slightly displaced to the left. The diaphragm is normal. There is no evidence of collapse of the lung on either side.

Here is the final film. This mass at the right lung root has increased in size and now involves the pleura, and the mass in the upper mediastinum has also enlarged. Here is the interlobar septum at about normal level. There is still no collapse of the lung. In this lateral view the mass lies anteriorly and extends into the upper lobe around the trachea.

These are the films of the gastrointestinal tract. The antrum of the stomach is contracted in these two films. I can see only what is already given in the report, that is spasm of the antrum of the stomach. The antrum is not filled. You cannot say that no intrinsic disease exists from that examination. Here in this film, however, it looks as if there were no intrinsic disease. The gallbladder is as described. I do not see any shadow at all. The spasm of the antrum probably due to extrinsic disease, and failure of function of the gallbladder, are all that I visualize.

Differential Diagnosis

Dr. Myles P. Baker: To summarize, this is a fairly rapidly fatal illness. A man of 53 develops out of the clear a paroxysmal right upper quadrant pain, possibly associated with change of position, such as forward bending; but noticeably unaffected by eating and not associated with gastrointestinal upsets of any form. There were nonspecific warnings that this process might be producing anemia of low grade and fever, but he worked until approximately seven weeks before his death in what I take to be a pronounced cachexia. Three weeks before entry the pain became a more insistent ache. There may have been jaundice at this time. The pain radiated through or up to both shoulders.

Physical examination revealed little except right upper quadrant tenderness and fever, with a slightly elevated leucocyte count. He was not icteric. Nothing was found to implicate the heart in the symptomatology and no hard prostate was felt, as with a malignant process.

During his first week in the hospital he continued to run a fever. This first x-ray revealed an enlarged hilum shadow on the right. There was evidence of possible chronic cholecystitis without stone demonstrable by x-ray. The significance of this enlarged hilum shadow remained in doubt.

They mention that the diastase index was normal. Dr. Crone tells me that the test has been done on several patients in the house in whom pancreatic disease was suspected; but it is of no value because you can have extensive pancreatic disease and get normal results from the test.

At the beginning of his second week he mentioned this right-sided chest pain with blood-streaked sputum and it was following that that the second x-ray revealed a rather rapidly enlarging mass in the right hilum. At this time he had developed an anemia out of proportion to the small amount of blood in his sputum. There was at this time evident myelocytosis, with an elevated serum phosphatase, a sign that the man had a rather marked osteoplastic process in his bones. This figure of 35 Bodansky units is in keeping with a metastatic malignancy involving bone. He remained subicteric and was, I take it, a problem in diagnosis on the ward. Two weeks later there is the note of a definitely enlarged liver and for the first time mention of a tender mass in the left upper quadrant. With this lead a pyelogram was done which revealed no kidney tumor. There was no evidence by x-ray of bone metastases. By x-ray there were signs of an extending process in the right lung field and hilum. Bronchoscopy, however, was negative. The fever, severe pain in his back, particularly in his later days, continued and death followed.

This man, it seems to me, certainly had either a bronchiogenic neoplasm, the first symptoms of which were due to retroperitoneal metastases, to lymph nodes in that region, or a growth originating in the retroperitoneal tissues, the pancreas or lymph nodes, with metastases to the mediastinal glands. The gastrointestinal tract is apparently intact by x-rays. The same arguments that Dr. Allen advanced hold true here.

For special comment I would like to mention first the character of this man's pain. I was interested to find that in discussions of series of cases of cancer of the body of the pancreas that paroxysmal pain was spoken of as not in-

frequently occurring. The French authors, such as Chauffard, are quoted as emphasizing the atrocious character of this paroxysmal pain, unaffected by eating, without nausea and vomiting, and mimicking tabetic crises. Whether this man had such fierce pain as that we do not know; at least, it was paroxysmal. I had not been aware that cancer of the pancreas led to such pain. It has been attributed to intimate relation between the body of the pancreas and the solar plexus. In about thirty cases of cancer of the pancreas which Kiefer reported three or four years ago four or five had this paroxysmal pain, later, as the course of the disease goes on, turning into an insistent boring pain, often in the right upper quadrant and therefore giving hope that one is dealing with gallbladder disease. The relief this man secured by standing up is said to be characteristic of retroperitoneal disease of this type. Some victims of cancer of the pancreas have been described as being only comfortable in the position of a Moslem at prayer toward Mecca. Others have this intractable sort of pain when lying down and are relieved when standing up. This is a nonspecific symptom, of course, and may be due to retroperitoneal lymphoma as well as to cancer of the pancreas.

Is it a possibility that we are dealing here with bronchiogenic carcinoma with retroperitoneal metastases? There have been cases discussed here in which the relatively small malignant growth at the hilum has really made itself manifest with metastases to the liver, giving the picture of liver failure, with jaundice and bringing up the differential diagnosis between carcinoma of the bile duct with metastases to the mediastinum, and bronchiogenic carcinoma with liver metastases. I think the rapid progress of this man's lesion in the hilar region, with its rapid growth out into the lung, would be rather more in favor of metastases from a retroperitoneal source rather than the manifestation of extension from a primary growth in the bronchus; and bronchoscopy revealed no growth.

Is it possible that we are dealing with lymphoma? Before this last case was presented I was about to say that—barring one case that I saw last winter—the absence of gastrointestinal symptoms was rather against it and more in favor of cancer of the pancreas, but here in the last case we have cancer of the pancreas with marked nausea and vomiting and duodenal obstruction. The instances of retroperitoneal lymphoma without enlargement of the peripheral lymph nodes that I have seen have happened to have more pain in the legs, perhaps edema of one leg and a mass that may be felt in the right lower quadrant as well as in the left upper quadrant, more signs of a diffuse process than this man showed.

How should we explain the blood picture? It seems to me more likely that this is a myelocytosis as the result of metastatic disease involving the bone marrow than that we are dealing with the most unlikely possibility of an aleukemic leukemia in this case, without the usual splenomegaly, or thrombopenic purpura.

We have had here in some of these cases for discussion a precedent for attributing left-sided backache such as this man had toward the end of his hospital stay to cancer of the pancreas. There have been cases in which there has been marked metastatic involvement of the mediastinal lymph nodes. Other cases of cancer of the body of the pancreas have not emphasized metastases to bone. Yet I am inclined to make cancer of the pancreas with metastases to the mediastinal lymph nodes and with metastasis also to bone not demonstrated by x-ray the first choice in this case, and to consider lymphoma as the second possibility and less likely.

Clinical Discussion

Dr. Chester M. Jones: The picture was that of metastatic malignancy. When he was on the service the question of course arose as to the source. I think that as we watched the case we felt a little more inclined to think it was primary in the lung than elsewhere in the body, but that is as far as we got toward making a flat diagnosis.

Clinical Diagnoses

Carcinomatosis, ? source.
Myelophthisic anemia.

Dr. Myles P. Baker's Diagnosis

Carcinoma of (body of) pancreas with metastases to the right hilus and to the bone marrow.

Anatomic Diagnoses

Carcinoma of the head of the pancreas with metastases to regional lymph nodes, retroperitoneal and mediastinal glands, liver, lungs, kidneys, adrenals, vertebrae and skull.
(Myelophthisic anemia).
Parathyroid hyperplasia, slight.

Pathologic Discussion

Dr. Benjamin Castleman: This man had a vertebral spine that was almost completely replaced by carcinoma. The only spots that are not cancerous are these isolated red spaces that you see here. The remainder, the white areas, are all cancer. The extremely diffuse involvement of the spine plus its osteosclerotic nature probably account for the lack of visualization on the x-ray film, for the pseudoleukemic blood findings, and also for the slight parathyroid

hyperplasia that is not uncommon in severe bone marrow replacement. The primary source of the cancer was in the head of the pancreas which was replaced by a firm irregular mass, 8 by 7 by 6 centimeters, which did not involve much of the body of the pancreas. Although metastatic glands surrounded the bile ducts they did not compress them sufficiently to produce obstruction and jaundice.

The liver and kidneys contained numerous metastatic nodules and both adrenals were almost completely replaced by tumor. The pleural surfaces of the lungs were studded with small metastases and there were also a few that involved the parenchyma. In the floor of the right middle fossa of the skull were two small tumor nodules. The most interesting metastases were those to the bronchial lymph nodes which produced a mass, 7 by 5 by 4 centimeters, that deflected the right bronchus superiorly and anteriorly and extended along the right posterior aspect of the trachea. There was, however, no perforation or significant obstruction of the bronchus, the latter accounting for the absence of lobar collapse on the x-ray film, a fact which helped Dr. Baker to eliminate a primary bronchiogenic tumor.

A few years ago we presented here a similar case in which the mediastinal metastases were so large that a diagnosis of mediastinal lymphoma was made clinically. Because of these two cases we thought it might be interesting to review all the autopsied cases of carcinoma of the pancreas during the past 36 years for distribution of metastases and other significant data.

We have had during this period 91 cases and it is noteworthy that, of the 84 in which the clinical diagnosis is recorded, the correct diagnosis was made antemortem in only 42, exactly 50 per cent. A palpable mass was noted in only 36 per cent of the cases. The likelihood of a correct diagnosis is obviously greatly influenced by the presence or absence of jaundice and this in turn is apt to depend upon the exact anatomic location of the tumor, tumors of the head being naturally frequently associated with jaundice and tumors of the body and tail rarely so. That a significant number of exceptions to this rule occur is shown, however, by the fact that, of 69 cases with involvement of the head of the organ, 5 were free from any trace of jaundice; whereas, of 21 cases where body or tail were involved without the head, jaundice was present in 4. Among these 21 cases of cancer of the body or tail, the correct diagnosis was made antemortem only twice.

That painless jaundice is not the rule in cancer of the pancreas was also clearly brought out. Pain was complained of in 70 per cent of the cases and pain in or radiating to the back was specifically mentioned in 25 per cent. In the small group of cases of the body and tail, back pain seemed proportionally a little more frequent, 33 per cent to be exact. The distribution of metastases is shown in the following table.

DISTRIBUTION OF METASTASES

(Present in two-thirds of the cases)

Liver	47	Gallbladder	5
Lymph Node	42	Kidney	4
Peritoneum	15	Colon	3
Lung	15	Pleura	3
Adrenal	9	Heart	3
Mediastinum	6	Bone	2

Brain, Uterus, Ovary, Stomach and Thyroid 1

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HOME NURSING

THE article on "Home Nursing" by Doctor Worcester,* raises a number of interesting questions, not the least important of which concerns the way to meet the need for adequate nursing in the home. That the need exists may, for the present, be taken for granted, and that it will persist for another generation at least is a not unreasonable expectation. How urgent the need may be is a question for some qualitative standard. Also the magnitude of the need must be measured by some readily accessible scale.

The argument starts from the fact that nursing in the home is too often unsatisfactory, and a multitude of grounds for dissatisfaction can be set forth by the combined efforts of physician, patient, patient's family and nurse. It then proceeds to the fact that the nurse has been trained in the hospital; and draws the conclusion that *therefore* she does not know how to nurse in the home. But do nurses enter homes for the first time when they are called there to nurse the sick? Have they not been brought up in homes? Well, perhaps not according to some descriptions of contemporary life.

In substance the charge is that the nurse trained in the hospital becomes institutionalized. But nurses are not the only persons in the community suffering from this condition. The complaint is indeed general and as to the nurse is justified if it is re-worded to read: "Too many nurses become institutionalized."

The solution of this problem lies at a deeper level than is generally reached in attempted analyses of the nursing situation. It is but a superficial explanation to say that the nurse in the hospital is too busy to learn the elaborate details which characterize the nursing by the finished artist. The specific defect in nurses, and in others in many walks in life is that the mind becomes rigid: there is lost the capacity for adaptation to changed circumstances.

It is possible to assume that a nurse cannot nurse successfully in a home unless she has received her training as a nurse in a home. It is in the case of most nurses an unwarranted assumption. Rather is it true that if she has had the right kind of training in the hospital, so that her innate capacity for adaptation is not brought to senescent rigidity in the three years of hospital life, she will be able to adjust herself to nursing in home conditions. That is, if she grew up in a home.

There is another reason why home nursing has remained in less favor with the nurse, in comparison with the "district and public health nursing". It is social: too often the nurse is regarded not merely as a domestic servant, in some communities this implies no degradation, but as a menial, in the contemptible sense.

There is another point often overlooked in making comparisons, namely, that the district nurse, in Boston formerly working under the Instructive District Nurses Association, not only nurses patients in their homes but also has an important function in instructing the family in the care of the patient.

The problem of adequate nursing in the home is a real problem, urgent and of signal magnitude, but its solution would seem to be not in creating new institutions but by improving and extending institutions already in existence. Some District Nursing Associations actually give to graduate nurses and to undergraduate nurses the training desired under adequate supervision. It is more fundamental, however, that the hospital school of nursing assist the pupil nurse in the truly educational work of postponing as long as possible the inevitable rigidity of the mind common enough in old age, but characteristic of the institutionalized person, irrespective of years.

*Page 1027 of this issue of the *Journal*.

THE REPORT OF THE MASSACHUSETTS BOARD OF REGISTRATION IN MEDICINE

THE report of the Massachusetts Board of Registration in Medicine for 1935 is at hand and is an interesting document because of the factual data contained therein and certain recommendations submitted.

The facts set forth substantiate the arguments, submitted to the legislature of 1936, respecting the large number of applicants who were inadequately prepared to enter upon the practice of medicine, as shown by 117 rejections of 159 examined in March, 138 of 216 in July, and 159 of 230 in November, an average percentage of rejections of over 70, or a total of 414 rejections of 609 applicants.

There were three special examinations of one applicant in each, probably to enable certain well-qualified applicants to engage in some essential field of practice in the state.

In addition to those registered by examination, 71 others were enrolled as legally qualified through certification by the National Board. This authority was conferred by the legislature in 1923 and is now the custom in forty-three states.

The records give the results of the examinations of the graduates of all medical schools from which the applicants came, and show to some extent the quality of instruction carried on by the several institutions. A significant question may be, Have those medical schools represented by the larger groups of rejected applicants shown good judgment in accepting students who are not naturally adapted to, or educationally qualified for, the study of medicine? Fortunately for many would-be doctors, the better equipped medical schools do not feel warranted in encouraging the wasted time devoted to medical study by those whose lives should be spent in other pursuits. Now that the Massachusetts law has conferred upon a board, authority to determine the educational standards of medical schools and to decline to accept applicants from nonapproved schools, there will be fewer rejections after 1939 when this law shall have become operative.

There is a very important fact set forth in the report which is that the work of the Board is a source of state revenue for, since its creation, it has earned for the Commonwealth $66,561.46 in excess of the costs of operation.

Since this is a public health measure designed to provide therapeutic and preventive service, it is regrettable that the earnings of the Board are not more generally available for the protection of the people against the practice of irregulars.

Under the financial system of the Commonwealth, the earnings of the Board of Registration in Medicine are not at the command of the Board, but are deposited with the Treasurer, and the expenses of the Board are defined in budgeting appropriations which have never been adequate for the needs of this department.

The Board believes that a careful census of the physicians of the state should be made at regular intervals in order to detect impostors. This custom prevails in many states with the possible result that irregular practitioners may migrate to other less vigilant sections of the country. A request for an appropriation for this study has been submitted to the legislature, but did not meet with the general approval of the medical profession and the appeal was denied. With the appropriation of a suitable sum from the earnings of the Board, this investigation could be undertaken without making a direct tax on the physicians and seems to be desirable. Since power has been given in 1936 to a commission to determine the standing of medical schools, any other steps designed to promote the quality of medical service are worthy of approval.

We urge careful study of existing conditions and the conferring of such added powers as may enable the Board of Registration in Medicine to serve the Commonwealth adequately.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

MYERSON, ABRAHAM. M.D. Tufts College Medical School 1908. Professor of Neurology, Tufts College Medical School. Clinical Professor of Psychiatry, Harvard University Medical School. Visiting Physician, Department of Nervous Diseases, Boston City and Beth Israel Hospitals. Division of Research, Boston State Hospital. Address: 475 Commonwealth Avenue, Boston, Mass. Associated with him are

RINKEL, MAX. M.D. University of Kiel (Germany) Medical School 1926. Formerly, Chief Assistant Physician, Psychiatric and Neurological Clinic of the Medical Academy, Düsseldorf, Germany. Now, Research Associate, Boston State Hospital. Address: Boston State Hospital, Dorchester Center, Mass. And

DAMESHEK, WILLIAM. M.D. Harvard University Medical School 1923. Associate Physician, Beth Israel Hospital. Assistant Professor of Medicine, Tufts College Medical School. Research Associate, Boston State Hospital. Address: 371 Commonwealth Avenue, Boston, Mass. Their subject is "The Autonomic Pharmacology of the Gastric Juices." Page 1005.

THORNDIKE, AUGUSTUS, JR. M.D. Harvard University Medical School 1921. F.A.C.S. Associate Surgeon, The Children's Hospital, Bos-

ton. Consulting Surgeon, Canton Hospital School. Surgeon, Milton Hospital and Department of Hygiene, Harvard College. Assistant in Surgery, Harvard University Medical School. Address: 319 Longwood Avenue, Boston, Mass Associated with him is

PIERCE, F. RICHARD. M.D. Harvard University Medical School 1934. Formerly, Intern, Surgical Service, The Children's Hospital, Boston. Now, Assistant Resident in Surgery, Henry Ford Hospital, Detroit, Mich. Address: Henry Ford Hospital, Detroit, Mich. Their subject is "Fractures in the Newborn. A Plea for Adequate Treatment." Page 1013.

STEINBERG, NAAMAN. M.D. Tufts College Medical School 1919. Proctologist, Beth Israel Hospital, Boston, Mass. His subject is "Recent Advances in the Treatment of Rectal Diseases by Injection Methods in Ambulatory Patients. II. Pruritus Ani." Page 1019. Address: 311 Commonwealth Avenue, Boston, Mass.

WILLIAMS, JOHN T. M.D. Harvard University Medical School 1904. F.A.C.S. Instructor in Gynecology, Harvard University Medical School. Surgeon-in-Chief for Gynecology and Obstetrics, Boston City Hospital. Obstetrician-in-Chief, Whidden Memorial Hospital, Everett; Chelsea Memorial Hospital, and Winthrop Community Hospital. His subject is "Epidemic Puerperal Sepsis." Page 1022. Address: 429 Beacon Street, Boston, Mass.

WORCESTER, ALFRED. A.M., Sc.D., M.D. Harvard University Medical School 1883. Henry K. Oliver Professor of Hygiene Emeritus, Harvard University. His subject is "Home Nursing." Page 1027. Address: 314 Bacon Street, Waltham, Mass.

The Massachusetts Medical Society

FOURTH ANNUAL POSTGRADUATE MEDICAL EXTENSION COURSE

The following sessions have been arranged by the Committee for the week beginning November 30:

Bristol North

Thursday, December 3, at 4:00 p. m., at the Morton Hospital, Taunton. Subject: *Complications* of Diabetes and Their Treatment. Coma; Insulin Reactions; Surgery (Gangrene, Carbuncle, Etc.); Marriage and Pregnancy; Tuberculosis and Heart Disease. Instructor: E. P. Joslin. Arthur R. Crandell, Chairman.

Bristol South (Fall River Section)

Monday, November 30, at 4:00 p. m., at the Stevens Clinic of the Union Hospital, Fall River. Subject: *Complications* of Diabetes and Their Treatment. Coma; Insulin Reactions; Surgery (Gangrene, Carbuncle, Etc.); Marriage and Pregnancy; Tuberculosis and Heart Disease. Instructor: Alexander Marble. Howard P. Sawyer, Co-Chairman.

Bristol South (New Bedford Section)

Friday, December 4, at 4:00 p. m., at St. Luke's Hospital, New Bedford. Subject: *Complications* of Diabetes and Their Treatment. Coma; Insulin Reactions; Surgery (Gangrene, Carbuncle, Etc.); Marriage and Pregnancy; Tuberculosis and Heart Disease. Instructor: H. F. Root. Robert H. Goodwin, Co-Chairman.

Middlesex North

Friday, December 4, at 7:00 p. m., at St. Joseph's Hospital, Merrimack Street, Lowell. Subject: Blood Diseases: The Hemoglobin and Red Blood Cells in Relation to Disease. Instructor: W. P. Murphy. Samuel A. Dibbins, Chairman.

Middlesex South

Tuesday, December 1, at 4:00 p. m., at the Cambridge Municipal Hospital, Cambridge. Subject: Acute Abdominal Emergencies. Instructor: A. W. Allen. Edmund H. Robbins, Chairman.

Norfolk South

Monday, November 30, at 8:30 p. m., at the Quincy City Hospital. Subject: Psychiatry: (a) Psychobiology in General Medicine. (b) The Common Neuroses. Instructor: H. C. Solomon. David L. Belding, Chairman.

Plymouth

Tuesday, December 1, at 4:00 p. m., at the Brockton Hospital, Brockton. Subject: Blood Diseases: The Hemoglobin and Red Blood Cells in Relation to Disease. Instructor: C. W. Heath. W. H. Pulsifer, Chairman.

Worcester North

Friday, December 4, at 4:30 p. m., at the Burbank Hospital, Fitchburg. Subject: Diabetes: General Plan of Treatment in *Uncomplicated* Cases; Diet; Insulin (Regular and Protamine); Exercise. Instructor: E. P. Joslin. Edward A. Adams, Chairman.

MISCELLANY

WALTER REED'S WIDOW HONORED

Amid a hushed and respectful audience the scientific epic of how Major Walter Reed of the U. S. army medical corps proved that yellow fever is transmitted by mosquitoes was retold recently. The recitation of the 36-year-old classic of medicine was provoked by the presentation to Mrs. Walter Reed, widow of the late army surgeon, of the Walter Reed medal by the American Society of Tropical Medicine.

Mrs. Reed was unable to attend the society's

luncheon and, instead, the medal was presented to Major General Walter L. Reed, son of Major and Mrs. Reed, now inspector general of the army.

At the same time a similar Walter Reed medal was presented to the Rockefeller Foundation, which has aided in the work of eradicating yellow fever in many parts of the world by applying the discovery of Major Reed. It was received by Raymond B. Fosdick, president of the foundation, for present workers in yellow fever eradication and those who have died in the work.—*Boston Herald*, November 20.

HEALTH OFFICERS' MONTHLY STATEMENT OF VENEREAL DISEASES REPORTED IN THE NEW ENGLAND STATES

SEPTEMBER, 1936

This statement is issued monthly for the information of health officers in order to furnish current data as to the prevalence of the venereal diseases. The following reports were received from State Health Officers. The figures are preliminary and subject to correction. It is hoped that this will stimulate more complete reporting of these diseases.

State	Syphilis		Gonorrhea	
	Cases Reported During Month	Monthly Case Rates per 10,000 Population	Cases Reported During Month	Monthly Case Rates per 10,000 Population
Connecticut	198	1.15	123	.72
Maine	44	.52	58	.69
Massachusetts	463	1.06	571	1.31
New Hampshire	14	.28	7	.14
Rhode Island	98	1.44	65	.95
Vermont	33	.88	32	.85

Treasury Department—U. S. Public Health Service.

CORRESPONDENCE

PREVENTIVE MEDICINE AND CURATIVE MEDICINE

Editor, *New England Journal of Medicine*,

With the extension of preventive medicine as organized by the various health departments, federal, state or local, with the taxpayers' money, it is important for both laymen and physicians to realize the difference between preventive medicine and curative medicine.

At the present time a part of curative medicine such as the treatment of mental disease, tuberculosis and cancer is paid for by the taxpayers' money and that part of it is not always available to all the citizens.

On the other hand, preventive medicine which is paid for by the taxpayers' money at the present time is available to practically all the citizens, rich or poor, if they choose to avail themselves of the opportunity.

Recently there has been a reasonable amount of complaint on the part of practitioners that abuse of charity exists in curative medicine and that encroachment is also made, by preventive medicine, upon the practice of physicians. These two complaints should be kept separate by the practitioners.

Attempts have been made by the District Societies of the Massachusetts Medical Society located in the City of Boston to eliminate, so far as possible, the abuse of charity in the treatment of disease in the large clinics. Although theoretical safeguards against this abuse have been established in all the clinics, there is probably still an appreciable amount of abuse which it is hoped as time goes on will be further eliminated.

These same District Societies have also studied the question of the encroachment of preventive medicine upon the work of the practitioners with the object of eliminating this encroachment if it seems justifiable. It must be realized that much of this preventive medicine is educational work and the people are slow to pay directly for something about the value of which they are uncertain. Therefore, this work must not be handicapped by the objection of expense. Also, it must be remembered that all the citizens are entitled to these preventive medicine procedures paid for by taxes according to the present view of those in authority. In order to keep some of this work, such as immunization and health examination in the hands of the practitioners, it is necessary to establish the fact that it is better for the patients to have this work done by their family physician. In this regard it is interesting to find that the Health Department of Boston through its Health Commissioner, Dr. William B. Keeler, and the Deputy Commissioner, Dr. Charles F. Wilinsky, instruct their nurses to make every possible effort to have the public use their family physicians for immunization work and the work done by the Well Baby Clinics, and to refer to the Health Department Clinics in Boston only such people as are unable to employ a family physician or do not seem inclined to do so. The health authorities instruct their nurses that it is better for the individuals to have this work done by their family physicians. It is to be hoped that the nurses of the Public School Association will be instructed as clearly in this regard as are the Public Health Nurses.

The writer of this article has recently had the opportunity to be present at a meeting at which the nurses of the Health Department of the City of Boston were instructed along these lines and was also given an opportunity to present the practitioners' point of view and he is convinced that the Health Officers of the City of Boston sincerely hope that individuals will have this work done by their family physicians. The writer took the opportunity to call the nurses' attention to the fact that some of the physicians in Boston are organizing a Public Health Hour or making special arrangements so that some of these preventive medicine procedures can be offered to their patients at a price commensurate with the patient's ability to pay even if at less than

the usual fees. The writer feels that practitioners should make every effort to see that their examinations of apparently healthy patients are as thorough as those offered by the Health Department Clinics.

CHANNING FROTHINGHAM, M.D.

November 17, 1936.

A VOTE PASSED BY THE BOSTON SOCIETY OF ANESTHETISTS

November 19, 1936.

Editor, *New England Journal of Medicine,*

At a special meeting of the Boston Society of Anesthetists, held November 6, the following vote was passed. This vote was taken, not only after full discussion with Doctors Faxon and Blaisdell, but also after a series of conferences with anesthetists from New York, Rochester, Washington, and other places held during the October Congress of Anesthetists at Philadelphia:

"Resolved that it is the sense of this society that the Hospital Prepayment Plan as at present drawn up would, if the anesthetists should be included, tend to reduce them toward the position of technicians, would tend to place them under the control of the hospitals and would set them apart from other branches of medicine; that these influences would tend to stop progress in anesthesia and to push the practice of anesthesia back to its status of a few decades ago, and would ultimately result in poorer service to the patient; and that for these reasons the society stands against being included in the contract as now proposed."

Very truly yours,

RUSSELL F. SHELDON, M.D., *Secretary.*

RECENT DEATHS

HALLOWELL—CLEMENT HOWARD HALLOWELL, M.D., a retired physician of Billerica, Massachusetts, died at his home, November 13, 1936.

Dr. Hallowell was born in 1854, graduated from the Colby College, and from the medical school of Boston University in 1879. He formerly practiced in Norwood, Massachusetts, before moving to Billerica several years ago.

His widow, Mrs. Blanche G. Hallowell, and a daughter, Mrs. John Cochrane, of Billerica, survive him.

McNEISH—ALEXANDER MCNEISH, M.D., of Leicester, Massachusetts, died at his winter home, at St. Petersburg, Florida, November 20, 1936. Dr. McNeish came to Massachusetts from Sussex, New Brunswick, and entered the Tufts College Medical School, graduating therefrom in 1896. He retired from membership in the Massachusetts Medical Society in 1934.

Dr. McNeish had served as Leicester school physician and as a member of the School Board. In addition he had taken active positions in other civic activities and was a member of the Congregational Church.

His widow, Mrs. Esther (White) McNeish, and a daughter, Miss Marion McNeish, survive him.

WILBUR—SARAH MANN WILBUR, M.D., of Springfield, Massachusetts, died at her home, November 20, 1936, after a practice of over forty years.

Dr. Wilbur was born in Westerly, R. I., in 1853, the daughter of Dr. William Hale Wilbur, and, after her graduation from Rutgers Female College, entered the Woman's Medical College of Pennsylvania, graduating therefrom in 1885. She had served at the New England Hospital for Women and Children, as resident physician at the Monson State Primary School, the Staten Island branch of the Nursery and Children's Hospital, and at the home for Friendless Women. In May 1888, she began practice in Springfield and devoted much of her work to obstetrics.

Dr. Wilbur was a member of the Springfield Women's Medical Society, a retired member of the Massachusetts and Rhode Island Medical Societies, and also of the American Medical Association.

Two nephews, John Wilbur, of New London, Connecticut, and William Hale Wilbur, of Hawaii, and a niece, Mrs. Edith Keller, of Newton Center, survive her.

NOTICES

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 p. m. on Thursday, December 3, in the Amphitheater of the Peter Bent Brigham Hospital, Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic, Harvard Medical School and Physician-in-Chief, Peter Bent Brigham Hospital, will give a medical clinic. To it are cordially invited practitioners and medical students.

LECTURE ON ARTHRITIS AT THE ROBERT BRECK BRIGHAM HOSPITAL

Dr. H. Warren Crowe, Director of the Rheumatism Clinic at Charterhouse, London, will be the guest speaker at a meeting on Monday, November 30, at 8 o'clock at the Robert B. Brigham Hospital.

Dr. Crowe will speak on the results of his work with vaccine in the treatment of arthritis over a period of twenty years.

Physicians and medical students are cordially invited.

APPOINTMENT OF DR. GODDU

Dr. Louis A. O. Goddu has recently been appointed Clinical Professor of Orthopedic Surgery at Tufts College Medical School.

REPORT AND NOTICES OF MEETINGS

OFFICERS OF THE NEW ENGLAND BRANCH OF THE AMERICAN UROLOGICAL ASSOCIATION

At a meeting of the New England Branch of the American Urological Association held in Boston, on

November 12, 1936, the following officers were elected for the ensuing year:

President—Fletcher H. Colby, M.D., Boston.

Secretary-Treasurer — George C. Prather, M.D., Boston.

Executive Committee — Samuel N. Vose, M.D., Chairman, Boston; W. O. Wilder, M.D., Springfield, Mass.; C. Harold Jameson, M.D., Rockland, Me.; John C. Eckels, M.D., Lisbon, N. H.; W. G. Townsend, M.D., Burlington, Vt.; Howard K. Turner, M.D., Providence, R. I.; C. H. Neuswanger, M.D., Waterbury, Conn.

GREATER BOSTON MEDICAL SOCIETY

The next meeting of the Greater Boston Medical Society will be held on Tuesday, December 1, at 8:30 p. m. at the Beth Israel Hospital Auditorium, Boston.

PROGRAM

Panel Discussion.

Abdominal Pain in Infancy and Childhood.

Chairman: Hyman Green, M.D.

Panel: Doctors J. Wallace, G. Rubin, M. H. J. Freedman, M. Ingall, and G. Kahn.

H. A. Kontoff, M.D., *President*,
D. B. Stearns, M.D., *Secretary*.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheater (Shattuck Street Entrance), Tuesday evening, December 8, at 8:15 p. m.

PROGRAM

Presentation of Cases.

The Physiology of the Breast. By Edwin B. Astwood, M.D., The Johns Hopkins Hospital, Baltimore, Maryland.

Medical students and physicians are cordially invited to attend.

Marshall N. Fulton, M.D., *Secretary*.

THE BOSTON DISPENSARY

25 Bennet Street

Clinical Staff Meeting

A luncheon meeting of the Clinical Staff of the Boston Dispensary will be held on Friday, November 27, 1936, in the Auditorium on the second floor of the Dispensary building at 12 o'clock noon.

At 12:30 p. m. Dr. Richard H. Norton, Chief of the Dental Department, will speak on "Correlation of Dentistry and Medicine."

An invitation is extended to any who are interested in the subject. Luncheon to nonmembers, 35 cents.

Oliver G. Tinkham, M.D., *President*,
Louis E. Wolfson, M.D., *Secretary*.

BOSTON PATHOLOGICAL SOCIETY

The next meeting of the Boston Pathological Society will be held in the Amphitheater of the Children's Hospital (Entrance on Vila Street), Tuesday, December 1, at 8 p. m.

PROGRAM

1. Chromosome Structure, with Relation to Radiation. Alfred Marshak, Ph.D.
2. The Effect of Radiation on the Skin. Shields Warren, M.D.

Physicians and medical students are invited to attend. The program will be followed by a business meeting of the Society.

Sidney C. Dalrymple, M.D., *Secretary*.

BOSTON UNIVERSITY MEDICAL SOCIETY

Meeting in the Evans Memorial Auditorium

78 East Concord Street, Boston

Monday Evening, November 30, 1936, at 8 o'clock

PROGRAM

Presentation of Case:

"Protamine Insulin and the Hagedorn Era of Diabetes"—Dr. Elliott P. Joslin, Dr. Priscilla White.

Medical students, nurses and physicians are cordially invited to attend.

Robert E. Moss, *Secretary*.

FAULKNER HOSPITAL CLINICAL MEETING

The next meeting will be held on Thursday afternoon, December 3, at 5:00 p. m. In addition to the usual clinical pathological conference, Dr. Arthur T. Hertig will talk on "Pathology of Abortion."

All physicians are invited.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, NOVEMBER 30, 1936

Monday, November 30—

*8 p. m. Robert Breck Brigham Hospital. Lecture on Arthritis.

8 p. m. Boston University Medical Society. Evans Memorial Auditorium, 78 East Concord Street, Boston.

Tuesday, December 1—

9:30 a. m. Massachusetts General Hospital. Thoracic Clinic. Ether Dome.

*8 p. m. Boston Pathological Society. Amphitheater of the Children's Hospital (entrance on Vila Street).

8:30 p. m. Greater Boston Medical Society. Beth Israel Hospital Auditorium.

Wednesday, December 2—

New England Obstetrical and Gynecological Society. University Club, Boston.

8 a. m. Massachusetts General Hospital. Grand Rounds, Orthopedic Department.

†12 m. Clinico-Pathological Conference. Children's Hospital Amphitheater.

4 p. m. - 5 p. m. Surgical Pathological Conference, Dr. Cutler and Dr. Wolbach, Peter Bent Brigham Hospital.

Thursday, December 3—

8 a. m. Massachusetts General Hospital, Circulatory Clinic.

*8:30 - 9:30 a. m. Exchange visit, Surgical and Orthopedic Staffs of the Peter Bent Brigham and the Children's Hospitals, held this week at the Children's Hospital.

11 a. m. Massachusetts General Hospital. Medical Grand Rounds.

12 m. Massachusetts General Hospital. Clinical-Pathologic Conference.

*3:30 p. m. Medical Clinic, Peter Bent Brigham Hospital. Dr. Henry A. Christian.

*5 p. m. Faulkner Hospital Clinical Meeting.

Friday, December 4—

10 a. m. Massachusetts General Hospital. Fracture Rounds.

Saturday, December 5—

*10 a. m. - 12 m. Staff Rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

November 27—The Boston Dispensary, Clinical Staff Meeting. See page 1047.

November 30—Lecture on Arthritis at the Robert Breck Brigham Hospital. See page 1046.

November 30—Pharmacopoeial Hearing in Washington, D. C. Hotel Washington at 10 a. m. and 2 p. m.

November 30—Boston University Medical Society. See page 1047.

December 1—Boston Pathological Society. See page 1047.

December 1—Greater Boston Medical Society. See page 1047.

December 1—Lawrence Cancer Clinic. See page 997, issue of November 19.

December 2—New England Obstetrical and Gynecological Society, University Club, Boston.

December 3—Medical Clinic, Peter Bent Brigham Hospital. See page 1046.

December 3—Faulkner Hospital Clinical Meeting. See page 1047.

December 3-5—Annual Conference of the National Society for the Prevention of Blindness, Columbus, Ohio.

December 7—Physicians and medical students are cordially invited to attend a clinic presented by the Medical, Surgical and Orthopedic Services of the Infants' and Children's Hospitals at 4 p. m. on the first Monday of each month, in the Amphitheater of the Children's Hospital.

December 8—Harvard Medical Society. See page 1047.

December 10—Pentucket Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, at 8:30 p. m.

December 11—William Harvey Society. Auditorium, Beth Israel Hospital, Boston, 8 p. m.

December 15—Massachusetts Eye and Ear Infirmary, Monthly Clinico-Pathological Conference. See page 949, issue of November 12.

February 25, 26, 27, 1937—The New England Hospital Association. Hotel Statler, Boston.

March 30 - April 2, 1937—First International Conference on Fever Therapy. Postponement notice. See page 52, issue of July 2.

April 21-24, 1937—American Society for Experimental Pathology. See page 1075, issue of May 21.

October 25-29, 1937—American College of Surgeons. Chicago, Illinois.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

December 2—Salem Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. John W. Strieder, Associate Professor of Chest Surgery, at Boston University School of Medicine. Subject: "Pulmonary Suppuration."

FRANKLIN DISTRICT MEDICAL SOCIETY

Will meet at the Weldon in Greenfield at 11 a. m. the second Tuesdays of January, March and May.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

January 13, 1937—Bear Hill Golf Club, Stoneham.

March 16, 1937—Danvers State Hospital, Danvers.

May 11, 1937—Bear Hill Golf Club, Stoneham.

KENNETH L. MACLACHLAN, M.D., Secretary.
1 Bellevue Avenue, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

January 19, 1937—8:15 p. m. The Peter Bent Brigham Hospital. Communications and Case Presentations by the Staff. Suggested title—"Abdominal Pain from the Medical and Surgical Standpoint." Details of program to be announced.

February 23, 1937—Time, place and details of program to be announced.

March 30, 1937—8:15 p. m. New England Deaconess Hospital. A Symposium on Diabetes entitled "A Survey of the Diabetic Work of the George F. Baker Clinic in the New England Deaconess Hospital." Communications and Case Presentations by the Staff. Drs. Elliott P. Joslin, Howard F. Root, Priscilla White, Alexander Marble and Allen P. Joslin.

May, 1937—Annual Meeting. Details to be announced.

Note: The Censors will meet for the examination of candidates on the first Thursday of May, 1937. Fee of $10.00 is payable at the time of examination. Application blanks may be obtained by writing the Secretary, furnishing name, address and name of school of graduation in medicine. Application must be made at least three weeks prior to date of examination. Candidates whose applications are on file will receive proper notices.

FRANK S. CRUICKSHANK, M.D., Secretary.
1247 Beacon Street, Brookline.

PLYMOUTH DISTRICT MEDICAL SOCIETY

January 21, 1937—11 a. m. Bridgewater State Farm.

March 18, 1937—11 a. m. Brockton Hospital

April 15, 1937—Annual Meeting. 11 a. m. Ducy Hospital.

May 20, 1937—11 a. m. Lakeville State Sanatorium.

FRED F. WEINER, M.D., Secretary.
231 Main Street, Brockton.

SUFFOLK DISTRICT MEDICAL SOCIETY

January 27, 1937—Boston Medical Library. 8:15 p. m. Joint Meeting with the Boston Medical Library. "Anthropology." Dr. Carleton S. Coon.

March 31, 1937—Boston Medical Library. 8:15 p. m. "Social Insurance—It Affects the Medical Profession." Dr. Charles E. Mongan. Discussion: Dr. Channing Frothingham.

April 28, 1937—Annual Meeting. Boston Medical Library. 8:15 p. m. "Problems in Surgical Diagnosis." Dr. Howard M. Clute.

CONRAD WESSELHOEFT, M.D., President,
CHARLES C. LUND, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

December 9—St. Vincent Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

January 13, 1937—Worcester City Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

February 10, 1937—Worcester State Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

March 10, 1937—The Memorial Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

April 14, 1937—Worcester Hahnemann Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

May 6, 1937—At 4:30 in the rooms of the Worcester Medical Library, Inc., at 34 Elm Street, Worcester, will be held the spring meeting of the Board of Censors.

Wednesday Afternoon and Evening, May 12, 1937—Annual Meeting. Time and place for this meeting will be announced in an early spring issue of the Journal.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

BOOK REVIEW

Urology in Women. A Handbook of Urinary Diseases in the Female Sex. E. Catherine Lewis. Second Edition. 100 pp. Baltimore: William Wood & Company. $2.25.

This is a monograph on the urologic conditions in women. As the author states in the preface, "It is not intended to be a complete treatise on urology but rather to emphasize certain points which have a particular application to women patients." A section each is devoted to urethra, bladder, ureter, and kidney. Such pathologic entities as vesicovaginal fistula, interstitial cystitis, and strictures of the ureter are well treated. Only a small section is devoted to pyelitis, and pyelonephritis of pregnancy.

On the whole the book is well written but nothing new is found in this monograph that cannot be found in any good text of urology or gynecology.